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CONTENTS OF PREVIOUS NUMBER

JULY, 1931. NUMBER 1

The Present Status of Plaut-Vincent's Infection. Voss Harrell, M.D., Detroit.

Ultraviolet Light in Tuberculosis of the Larynx. Benjamin Katz, M.D., Los Angeles.

Carcinoma of the Sphenoid Sinuses with Invasion of the Cranial Cavity. Charles Davison, M.D., and Sidney Kahr, M.D., New York.

Osteoma of the Nasal Septum, with a Large Epidural Abscess; Operation; Recovery. Henry M. Goodyear, M.D., Cincinnati.

Radical Operation on the Frontal Sinus Under Local Anesthesia. Matthew S. Ersner, M.D., Philadelphia, and Joel Jay Pressman, M.D., Los Angeles.

Paranasal Sinusitis: Local Immunization Treatment. K. W. Amano, M.D., Philadelphia.

The Problem of Deafness. George E. Shambaugh, M.D., Chicago.

The Interpretation of Conduction Deafness: Report of Two Unusual Cases. A. G. Pohlman, M.D., St. Louis.

Physical Therapy in the Treatment of Diseases of the Ear, Nose and Throat. T. H. Odeneal, M.D., Beverly, Mass.

Calcigerous Metaplasia of the Auricular Cartilage. David R. Higbee, M.D., San Diego, Calif.

Clinical Notes:

- A New Bipolar Electrode for Coagulation of the Tonsils. Einar A. Sunde, M.D., Brooklyn.
- A Scouring Apparatus for the Cleansing of Cavities, such as the Paranasal Air Cells. Charles B. Hollis, M.D., Philadelphia.
- An Improved Curet for Operation on the Mastoid. Charles Nelson Spratt, M.D., Minneapolis.

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology for 1930: Progressive Deafness, Otosclerosis and Diseases of the Labyrinth. J. K. M. Dickie, M.D., Ottawa, Canada.

Abstracts from Current Literature.

Society Transactions:

- College of Physicians of Philadelphia, Section on Otolaryngology.

Directory of Otolaryngologic Societies.

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THE SPECIFIC PROPHYLAXIS OF PNEUMOCOCCUS AND STREPTOCOCCUS MENINGITIS

I. SERUM PROPHYLAXIS *

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While the incidence of pneumococcus and streptococcus meningitis is fortunately low, present methods of treatment are so unsatisfactory and the mortality so near 100 per cent, despite newer methods of treatment,[1] that it would appear worth while to inquire into the possibility of developing methods for the specific prophylaxis for these infections.

Unquestionably, adequate surgical drainage of suppurative foci in the middle ear, the mastoid and the nasal accessory sinuses is of importance in this respect, although in a large percentage of cases, meningitis develops after operations on infected sinuses and other foci because of a breaking down of chronic inflammatory barriers and a stimulation of infection.

Since it is usually possible to determine promptly and before operation the bacteria producing infections of the sinuses and the middle

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* Thesis presented by Dr. K. W. Amano to the Faculty in Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Doctor of Medical Science.

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ear and to include bacteriologic examination at the time of operation on the mastoid cells, the question arises whether or not it is possible and advisable to institute specific serum or chemotherapeutic prophylactic measures before or within a few days after operation in the case of infection caused by pneumococci and streptococci? Chronic infections of the middle ear and sinuses are not infrequently due to staphylococci, *B. Friedländer, B. pyocyaneus* and other organisms, but apparently these organisms are of much lesser importance in relation to the occurrence of diffuse septic meningitis.

The interest of one of us (Dr. Kolmer) in prophylaxis in pneumococcus meningitis dates back to 1920, when Kolmer and Idzumi [2] first showed that intraspinal injection into rabbits of ethylhydrocupreine hydrochloride alone or in combination with antipneumococcus serum had prophylactic effects when it was administered within from four to six hours after infection of the meninges with virulent type I pneumococci.

The results of extensive experiments performed during the past year with the lower animals indicate that it may be possible to prevent pneumococcus and streptococcus meningitis as a complication secondary to infections of the sinuses, middle ear and mastoid. It is our purpose in this paper to summarize our results briefly, to make an earnest plea for more frequent and more accurate bacteriologic studies before operation, and to suggest methods of serotherapeutic and chemotherapeutic prophylaxis applicable to human beings.

It is possible that the administration of autogenous vaccines of pneumococci and streptococci over a period of weeks may have some prophylactic value in raising the immunologic resistance of patients before operation, thereby possibly reducing the chances of extension of infection to the meninges. Our main interest in the present investigation, however, was to make a study of the possibilities of conferring immediate or almost immediate protection by means of serochemotherapeutic procedures performed at or soon after operation.

METHODS AND RESULTS OF PROPHYLAXIS IN PNEUMOCOCCUS MENINGITIS

Our first experiments were conducted with monkeys with the hope of establishing pneumococcus and streptococcus infections of the middle and inner portions of the ear and mastoid with gradual extension to the meninges, in order to duplicate as nearly as possible the conditions usually surrounding the production of meningitis in human beings. But repeated intratympanal inoculation of virulent type I pneumococci

2. Kolmer, J. A., and Idzumi, G.: Chemotherapeutic Studies with Ethylhydrocupreine and Mercurophen in Experimental Pneumococcus Meningitis of Rabbits, J. Infect. Dis. **26**:355 (May) 1920.

and hemolytic strains of streptococci into monkeys produced only purulent otitis media.

We then inoculated young broth cultures of virulent type I pneumococci into monkeys by the intracisternal route according to the method previously developed for producing experimental meningitis of dogs and rabbits.[3] This route of infection produced such fulminating types of meningitis as to cause the death of an untreated control in twenty hours with all of the usual signs and symptoms of acute meningitis.

But a monkey receiving an intravenous injection of 0.5 cc. of Felton's concentrated antipneumococcus serum per kilogram of body weight immediately after infection, followed by four intramuscular injections administered at daily intervals in the same dosage, survived for five days and fifteen hours, which, in view of the extremely fulminant meningitis of the control, indicated that this serum undoubtedly had protective power.

Our next experiment was conducted with dogs, but again we were unsuccessful in producing meningitis by inoculation of virulent type I pneumococci into the middle ear. Repeated injections of relatively large amounts of virulent pneumococci and streptococci produced acute otitis media, but clinical symptoms or cerebrospinal fluid signs of meningitis were not noted over several weeks of observation.

However, dogs inoculated intracisternally developed acute meningitis and succumbed in from one to five days. When 0.5 cc. of Felton's antipneumococcus serum per kilogram of body weight was given intravenously immediately after infection and was followed by two similar doses at daily intervals, about one half of the animals escaped meningitis; the others succumbed in from four to five days. However, three doses of 1 cc. each per kilogram given at daily intervals saved a larger percentage of animals.

We then tried single doses of from 0.2 to 0.4 cc. of the serum per kilogram of weight administered intravenously and 0.5 cc. administered intramuscularly, with single doses of ethylhydrocupreine hydrochloride given intravenously and intramuscularly and ethylhydrocupreine base administered by stomach tube. We were unable to ward off meningitis; the results of an experiment are summarized in table 1.

Fortunately injection of virulent pneumococci into the mastoid cells of rabbits could produce meningitis with a somewhat less rapid course than that following intracisternal injections of culture and therefore more suitable for our prophylactic experiments. Almost invariably, however, cultures of the heart's blood showed the presence of pneumo-

3. Kolmer (footnote 1, first reference).

cocci, so that in practically each instance death was due to meningitis and an associated septicemia.

The intravenous and intramuscular administration of relatively large doses of Felton's serum (1 cc. per kilogram) immediately after infection saved the majority of animals, and among those succumbing to the infection, life was prolonged several days beyond the survival of the

TABLE 1.—*Prophylaxis in Type I Pneumococcus Meningitis of Dogs (Intracisternal Inoculation)*

	Serum*		Ethyl Hydrocuprein†		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.5	Vein (3 doses)	0	0	50	4½ days	50
2	1.0	Vein (3 doses)	0	0	0	0	100
2	0.4	Vein (1 dose)	0.00125	Vein (1 dose)	100	4½ days	0
2	0.2	Vein (1 dose)	0.03	Stomach (1 dose)	100	4 days	0
2	0.5	Muscle (1 dose)	0.00125	Muscle (1 dose)	100	7½ days	0
2	Controls	0	0	0	100	1-5 days	0

* Felton's concentrated antipneumococcus serum.
† Ethylhydrocupreine hydrochloride was administered intravenously and intramuscularly; ethylhydrocupreine base by stomach tube.

TABLE 2.—*Serum Prophylaxis in Type I Pneumococcus Meningitis of Rabbits (Intratympanal Inoculation)*

	Serum*		Fatalities		
Number	Cc. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
4	1.0	Vein (1 dose)	50	10 days	50
3	1.0	Vein (2 doses)	0	0	100
3	1.0	Vein (3 doses)	0	0	100
3	1.0	Muscle (1 dose)	0	0	100
3	1.0	Muscle (2 doses)	0	0	100
3	1.0	Muscle (3 doses)	0	0	100
4	Controls	.....	100	2 to 3 days	0

* Felton's concentrated antipneumococcus serum.

untreated controls. In about half of the animals meningitis and septicemia developed after single doses, but almost all survived when second and third doses were given at daily intervals. The result of an experiment is summarized in table 2.

Equally encouraging results were observed with rabbits infected by the intratympanal injection of type I pneumococci and treated immediately after inoculation with intravenous and intramuscular injections of antipneumococcus serum prepared by the Massachusetts Board of Health Laboratories. The development of meningitis and associated

septicemia by this route of infection was sufficiently slow or delayed to serve excellently the purpose of the experiment, and single doses as small as from 0.2 to 1.0 cc. per kilogram administered intravenously and from 0.5 to 1.0 cc. per kilogram administered intramuscularly usually saved the animals, while untreated controls succumbed in about five days, as shown by the results of one experiment summarized in table 3.

However, ethylhydrocupreine hydrochloride administered intravenously and intramuscularly and ethylhydrocupreine base by stomach tube

TABLE 3.—*Serum Prophylaxis in Type I Pneumococcus Meningitis in Rabbits (Intratympanal Inoculation)*

	Serum*		Fatalities		
Number	Cc. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.2	Vein	0	0	100
2	0.5	Vein	0	0	100
2	1.0	Vein	0	0	100
2	0.5	Muscle	0	0	100
2	1.0	Muscle	0	0	100
2	Controls	0	100	4 to 6 days	0

* Massachusetts Board of Health serum.

TABLE 4.—*Ethylhydrocuprein Prophylaxis in Type I Pneumococcus Meninigitis in Rabbits (Intratympanal Inoculation)*

				Fatalities		
Number	Gm. per Kg.	Route	Number of Doses	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.0025	Vein*	1	100	3½ days	0
2	0.007	Vein*	3	100	2–5 days	0
2	0.015	Muscle*	3	100	3 days	0
2	0.025	Stomach†	3	100	3½ days	0
2	0.03	Stomach†	2	100	2¼ days	0
2	Controls	0	0	100	2½ days	0

* Ethylhydrocupreine hydrochloride.
† Ethylhydrocupreine base.

were without more appreciable prophylactic effects than a possible prolongation of life by one or two days, as shown in an experiment summarized in table 4. The doses given were approximately from ten to twenty times less than the maximum tolerated doses per kilogram of body weight. The first dose was administered immediately after infection and was followed by from two to three subsequent doses at daily intervals, but this did not prevent the onset of fatal meningitis and its associated septicemia. However, at the time the experiments were being conducted our culture possessed extreme virulence, so that the untreated controls succumbed in from two to three days.

It may be, however, that the administration of ethylhydrocupreine intravenously, intramuscularly or by stomach enhances the prophylactic activity of antipneumococcus serum, as indicated by the results of an experiment recorded in table 5.

For example, the administration of serum alone in a dose of 0.2 cc. per kilogram by intravenous and intramuscular injection usually did not save more than about one half of the animals, whereas when this treatment was combined with the oral administration of about 0.03 Gm.

TABLE 5.—*Prophylaxis in Type I Pneumococcus Meningitis in Rabbits (Intratympanal Inoculation)*

	Serum*		Ethyl Hydrocuprein†		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
3	0.2	Vein	0.03	Stomach (5 doses)	0	0	100
3	0.2	Muscle	0.03	Stomach (5 doses)	0	0	75
4	0.3	Muscle	0.025	Stomach (1 dose)	50%	4½ days	50
4	0.4	Muscle	0.0025	Muscle (1 dose)	50%	4½ days	50
3	1.0	Vein (3 doses)	0.0025	Vein (3 doses)	0	0	100
3	1.0	Vein (2 doses)	0.0025	Vein (2 doses)	0	0	100
3	1.0	Vein (1 dose)	0.0025	Vein (1 dose)	0	0	100
2	1.0	Muscle (3 doses)	0.0025	Muscle (3 doses)	0	0–	100
2	1.0	Muscle (2 doses)	0.0025	Muscle (2 doses)	0	0	100
2	1.0	Muscle (1 dose)	0.0025	Muscle (1 dose)	0	0	100
4	0.2	Vein	.....	.....	0	0	50
4	0.2	Muscle	.....	.....	50	2½ days	50
4	0.4	Muscle	.....	.....	..	.....	100
10	Controls	.....	.....	.....	100	2½ to 4½ days	0

* Felton's concentrated antipneumococcus serum.
† Ethylhydrocupreine hydrochloride was administered intravenously and intramuscularly; ethylhydrocupreine base, in suspension, by stomach tube.

of ethylhydrocupreine base each day for five days, the percentage of survivals was nearly 100 per cent. Undoubtedly, however, the maximum protection was afforded by the serum—and it was impossible to elicit any clearcut evidence of the prophylactic power of the ethylhydrocupreine as administered by these routes, although it is highly probable that its administration was at least helpful in some instances.

Intracisternal injections of virulent type I pneumococci usually produced severe fulminating meningitis with septicemia that was fatal in from two to three days, although we endeavored to inject minimal amounts of culture, with the result that some untreated controls survived indefinitely.

In these experiments, therefore, the prophylactic power of the serum alone administered by the intravenous or the intramuscular route in single doses of from 0.2 to 1 cc. per kilogram immediately after infection did not result in as high a percentage of survivals as in the experiments previously summarized, in which the pneumococci were inoculated intratympanally. As a general rule, the percentage of survivals was about 50 per cent, or about one-half less; this is doubtless because

TABLE 6.—*Prophylaxis in Type I Pneumococcus Meningitis of Rabbits (Intracisternal Inoculation)*

	Serum*		Ethylhydrocupreine Hydrochloride		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.2	Vein	0.00125	Muscle	100	6½ days	0
2	0.5	Vein	0.0025	Muscle	50	6 days	50
2	0.2	Muscle	0.00125	Muscle	100	6½ days	0
2	0.5	Muscle	0.0025	Muscle	50	3½ days	50
4	0.2	Vein	.....	.....	100	4½ days	0
4	0.5	Vein	.....	.....	50	1½ days	50
4	1.0	Vein	.....	.....	50	5½ days	50
4	0.2	Muscle	.....	.....	50	2½ days	50
4	0.5	Muscle	.....	.....	50	4½ days	50
4	1.0	Muscle	.....	.....	25	6½ days	75
8	Controls	.....	.....	.....	75	2½ days	25

* Felton's concentrated antipneumococcus serum.

TABLE 7.—*Prophylaxis in Type I Pneumococcus Meningitis of Rabbits (Intracisternal Inoculation)*

	Serum*		Ethylhydrocupreine Hydrochloride		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.2	Vein	0	0	100	3 days	0
2	0.4	Vein	0	0	50	4½ days	50
2	0.2	Muscle	0	0	100	1 day	0
2	0.4	Muscle	0	0	100	1½ days	0
2	0.2	Vein	0.0025	Muscle	100	1 day	0
2	0.4	Vein	0.0025	Muscle	50	3½ days	50
2	0.4	Muscle	0.0025	Muscle	50	3½ days	50
3	Controls	0	0	0	100	9 hours	0

* From the Massachusetts Board of Health.

intracisternal inoculation usually resulted in producing a more severe and more rapid and fulminating type of infection. But among the fatalities it was usually apparent that the serum alone or serum and the intramuscular injection of from 0.00125 to 0.0025 Gm. of ethylhydrocupreine hydrochloride appreciably prolonged the lives of many animals beyond the duration of the lives of the controls, as shown in the results of one experiment summarized in table 6.

In an effort to bring out more clearly the possible prophylactic activity of serum alone and serum with ethylhydrocupreine hydro-

chloride, rabbits were inoculated with larger amounts of culture, so that untreated controls succumbed in less than twenty-four hours, usually as the result of severe septicemia. In these experiments, the intravenous injection of 0.2 cc. amounts of serum per kilogram was without effect, although 0.4 cc. doses saved about one half of the animals. The intramuscular injection of 0.2 cc. of serum per kilogram along with the intramuscular injection of 0.0025 Gm. of ethylhydrocupreine hydrochloride per kilogram was likewise without appreciable effects; however, while the intramuscular injection of 0.4 cc. of serum per kilogram was without effect, when it was given along with an intramuscular injection of ethylhydrocupreine some of the animals survived, as shown in the results of an experiment summarized in table 7.

At this writing only few experiments have been conducted with types II and III pneumococci. Owing to the smaller amounts of anti-

TABLE 8.—*Prophylaxis in Type II Pneumococcus Meningitis of Rabbits (Intracisternal Inoculation)*

	Serum*		Ethylhydrocupreine Base		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.2	Vein	.....	.....	100	2 days	0
2	0.4	Vein	.....	.....	100	2-3 days	0
2	0.4	Muscle	.....	.....	100	2 days	0
2	0.4	Vein	0.03	Stomach (2 doses)	100	2½ days	0
2	0.4	Muscle	0.03	Stomach (3 doses)	100	2-3 days	0
2	Controls	.....	.....	.....	100	1-2 days	0

* Felton's concentrated antipneumococcus serum.

body present in serum for these types, especially for type III, less encouraging results were expected.

As shown in tables 8 and 9, the intracisternal injection of these strains into rabbits has produced severe infections, the resulting meningitis and septicemia producing fatal infection in all of the untreated controls in forty-eight hours or less.

It may be that the intravenous or intramuscular injection of the serum alone or in combination with ethylhydrocupreine base by stomach tube prolongs the lives of infected animals, but so far it has not resulted in preventing fatal infections, as shown in the summaries of two experiments in tables 8 and 9.

We have reason to state, however, that in intratympanal infections, the intravenous or intramuscular injection of serum in doses from two to four times larger than those employed for the type I infections causes some prophylactic activity, doubtless because the infections have been somewhat less fulminant. Larger doses of serum, however, are

required because of the lower antibody content, especially in virulent type III infections. The simultaneous administration of ethylhydrocupreine hydrochloride by intravenous or intramuscular injection may be helpful, although the evidence was much less definite when ethylhydrocupreine base was given by stomach tube.

METHODS AND RESULTS OF PROPHYLAXIS IN STREPTOCOCCUS MENINGITIS

Considerable difficulty was experienced in the production of streptococcus meningitis. As previously stated, monkeys and dogs inoculated even intracisternally with a culture of hemolytic streptococcus virulent for mice and rabbits did not develop meningitis and therefore could not be used in these experiments.

TABLE 9.—*Prophylaxis in Type III Pneumococcus Meningitis in Rabbits (Intracisternal Inoculation)*

	Serum*		Ethylhydrocupreine Base		Fatalities		
Number	Cc. per Kg.	Route	Gm. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.4	Vein	0	0	100	1–2 days	0
2	0.6	Vein	0	0	100	1½ days	0
2	0.6	Muscle	0	0	100	1½ days	0
2	0.6	Vein	0.03	Stomach (5 doses)	100	5 days	0
2	0.6	Muscle	0.03	Stomach (2 doses)	100	2 days	0
2	Controls	0	0	0	100	1½ days	0

* Felton's concentrated antipneumococcus serum.

Rabbits given intratympanal inoculations developed meningitis so irregularly that this route of infection could not be employed, although intracisternal injections resulted in producing infections that were usually fatal in from one to three days. Death, however, was apparently mostly due to associated septicemia, as cultures of the blood of the heart were invariably positive and often without definite macroscopic evidences of meningitis.

While intravenous and intramuscular injection of the scarlet fever antitoxins, chosen because of their antibody content for hemolytic streptococcus is higher than ordinary polyvalent antistreptococcus serums, even though the latter were not derived from patients with scarlet fever, prolonged the lives of most of the animals, yet none survived indefinitely, as shown in the results of one experiment summarized in table 10.

Owing to the difficulty of obtaining streptococci of sufficient virulence for these experiments and of maintaining their virulence, we have

not as yet been able to conduct a sufficient number of experiments to warrant more definite statements at present, although on the basis of experiments conducted up to the present time, we believe that the intravenous and intramuscular injection of antistreptococcus serum and especially of scarlet fever antitoxins, may possess some degree of prophylactic activity. Unfortunately, however, no chemotherapeutic compound has yet been found to improve the results materially, as the administration of the dyes, like acriflavine and gentian violet, and the mercurial compounds, like mercurophen and metaphen, do not appear to have improved the results.

CLINICAL RECOMMENDATIONS

We believe that the results of these experiments indicate that it may be possible to prevent pneumococcus meningitis following oper-

TABLE 10.—*Serum Prophylaxis in Streptococcus Meningitis in Rabbits (Intracisternal Inoculation)*

	Serum*		Fatalities		
Number	Cc. per Kg.	Route	Per Cent	Average Duration of Life	Survivals, per Cent
2	0.2	Vein	100	1-2½ days	0
2	0.5	Vein	100	3-5½ days	0
2	1.0	Vein	100	2-5¼ days	0
2	0.2	Muscle	100	1-2½ days	0
2	0.5	Muscle	100	1-2½ days	0
2	1.0	Muscle	100	2-4½ days	0
2	Controls	0	0	1½-2½ days	0

* Scarlet fever antitoxin.

ations on patients with pneumococcus infections of the frontal, ethmoid, sphenoid and other sinuses and mastoid cells by the administration of antipneumoccus serum, with or without the administration of ethylhydrocupreine, especially in infections due to types I and II pneumococci. Unfortunately, the outlook in streptococcus infections is not so good, because there is a lack of antistreptococcus serums comparable in therapeutic activity to the newer concentrated antipneumococcus serums, and because there is no chemotherapeutic agent for pneumococcus infections of which we are as hopeful of obtaining good results as we are of ethylhydrocupreine.

Fortunately, meningitis develops in only a small percentage of cases of infection of the sinuses and mastoids. It is hoped that clinical experience and knowledge have arrived at the point at which certain types of infection are known to be particularly dangerous from the standpoint of meningitis, and that at least some attempt will be made to protect patients from them.

At least, it is usually possible to ascertain before operation the etiologic nature of chronic infections of the sinuses and otitis media, and it is always possible to make smears and cultures of mastoid cells at the time of operation and thereby ascertain the nature of the infection within from twenty-four to forty-eight hours thereafter. In this connection it is proper to place emphasis on the necessity of using blood agar or brain hormone dextrose broth (the latter preferred) for making cultures, as streptococci or pneumococci may fail to develop on plain agar, and this may readily lead to error.

Not infrequently, cultures of pus from infected sinuses and middle ears show the presence of staphylococci in pure culture, and these organisms are very much less likely to result in meningitis, but this conclusion as to the nature of the infection is acceptable only when the cultures have been properly made on either of the aforementioned enriched mediums or on suitable substitutes by an otorhinologist.

If pneumococci are found, it is advisable to determine the type, which requires an additional eighteen to twenty-four hours, because this information is of value from the standpoint of dosage of serum and prognosis.

We believe that in severe infections with type I pneumococcus it is advisable to give an intramuscular injection of 10 cc. of Felton's concentrated antipneumococcus serum just before or immediately after operation and a similar dose the following day and a third dose about four days thereafter. In infections with a type II organism the dose may be the same or somewhat larger (like 15 cc.), and in type III infections at least 20 cc. should be given, since the serum contains less antibody than for type I and type II pneumococci. In infections with a group IV pneumococcus it may be advisable to give doses of 10 cc. even though the serum may contain no demonstrable amounts of antibody or much less than for the other types, in view of the encouraging results observed in the treatment for group IV lobar pneumonia.

We have been surprised to learn that the protection given rabbits by intravenous and intramuscular injections of the two antipneumococcus serums employed in this study may persist for many weeks, as determined by reinoculation of recovered animals at intervals by intratympanal injections of type I pneumococci. It is reasonable therefore to assume that protective amounts of antibody may persist in human beings over periods of from one to several weeks, during which recovery and walling-off of infected areas should reach the stage at which secondary meningitis may be prevented. For example, of 29 rabbits infected intratympanally with type I pneumococci and given prophylactic injections of serum alone or serum and ethylhydrocupreine, 25, or 86 per cent, survived. The 25 surviving animals were infected a second

time about two to three weeks later and 19, or 76 per cent, survived, while 5 of 7 controls succumbed. About two weeks later, the 19 survivors were infected a third time and all survived, while 2 of 5 controls succumbed. About two weeks later, the 19 survivors were infected a fourth time and all survived, while 2 of 4 controls succumbed. About two weeks later, the 19 survivors were infected a fifth time, and 18, or 95 per cent, survived, while 5 of 6 controls succumbed. From one to two weeks later, 18 survivors were infected a sixth time and all survived, while 3 of 4 controls succumbed. From one to two weeks later, 14 of the survivors were reinoculated and all survived, while 1 of 2 controls died. About two weeks later, the 14 survivors were

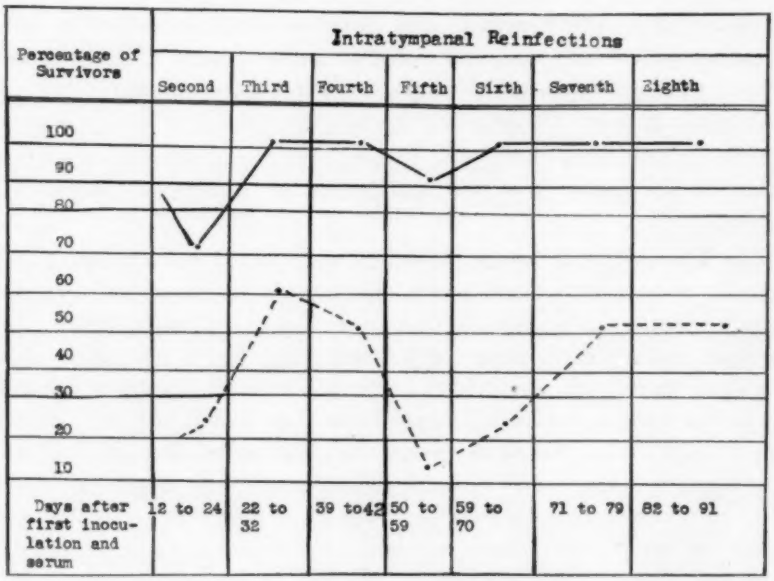

Chart showing the duration of acquired immunity in rabbits following the administration of antipneumococcus serum and repeated intratympanal inoculations of type I pneumococcus. The unbroken line indicates the percentage of survivals among rabbits receiving repeated intratympanal inoculations of type I pneumococci at intervals after the first or initial infection and administration of antipneumococcus serum; the broken line, the percentage of survivals of control rabbits receiving intratympanal inoculations of pneumococci at the same time.

reinoculated for the eighth and last time and all survived, while 2 of 4 controls succumbed (chart 1).

It is hardly likely that the high degree of immunity of the surviving animals was due to the persistence of antibody over so long a period (about one hundred and ten days), and that it was due in part to active immunization or vaccination resulting from the intratympanal inocu-

lations of pneumococci, but the results are indicative of an acquired immunity and are being studied further for a later and more detailed report.

Whether or not the administration of ethylhydrocupreine may prove to be an additional factor of practical value is largely a subject of conjecture so far as the results of these experiments are concerned. But it would appear from the experiments of Kolmer and Borow [4] that sufficient ethylhydrocupreine may be absorbed from the gastro-intestinal tract to raise appreciably the pneumococcidal activity of the blood, and since the outcome of some of our experiments was encouraging also from this standpoint, we believe that it is advisable to include the oral administration of 5 grains (0.324) Gm.) of ethylhydrocupreine base with milk every five hours during the first three days after operation. This dose amounts to about 25 grains (1.62 Gm.) of ethylhydrocupreine base per twenty-four hours for an adult and is without danger of producing toxic amblyopia except in those rarely found persons who have a natural allergic hypersensitiveness to quinine. Children may receive less ethylhydrocupreine according to weight, but the dosage of serum advised may be the same for children of 10 years or older and about half of that for younger children.

We make the following recommendations:

1. A thorough bacteriologic examination should be made of the pus from infected sinuses before operation and of the pus from the mastoid at operation.

2. In infections of adults with type I pneumococcus, 10 cc. of Felton's concentrated antipneumococcus serum should be injected intramuscularly just before or immediately after operation; a second dose should be given the following day and a third dose four days later. In infections with pneumococcus type II and group IV, the same dosage or 15 cc. should be used; in infections with type III pneumococcus, a dosage of 20 cc. should be employed.

3. The same doses should be given to children over 10 years of age; half doses to younger children.

4. In infections with the pneumococcus, 5 grains (0.324 Gm.) ethyl hydrochloride with milk should be given every five hours for three days after operation; children require less, according to weight.

5. In infections with the streptococcus, a therapeutic syringeful of a scarlet fever antitoxin should be given just before or immediately after operation, repeated the following day and again four days later (in unusually severe infections), or an intramuscular injection of concentrated polyvalent antistreptococcus serum should be given for the first dose followed by one or two doses at the same intervals. Children under 10 years require about one half of these amounts.

It is true that the majority of persons operated on for pneumococcus and streptococcus sinusitis and mastoiditis would escape meningitis with-

4. Kolmer, J. A., and Borow, L.: The Adaptation of the Heist-Lacy Method for Determining the Bactericidal Activity of Whole Blood for Chemotherapeutic Investigations, J. Infect. Dis. **31**:116, 1922.

out any of these prophylactic measures, and that it is always inadvisable to administer a serum if it can be safely avoided in order to avoid unnecessary allergic sensitization to horse serum. We believe, however, that in a percentage of cases, and especially in unusually extensive infections of the ethmoid and frontal sinuses and mastoid cells, these prophylactic measures are warranted, and that the results of our animal experiments indicate that a sufficient degree of protection may be afforded to tide persons over the critical periods following surgical procedures.

CONCLUSIONS

1. The intravenous and intramuscular administration of newer concentrated antipneumococcus serums to monkeys and dogs after intracisternal inoculations of virulent type I pneumococci had some prophylactic value in the prevention of meningitis.

2. The intravenous and intramuscular administration of those antipneumococcus serums to rabbits after inoculation of virulent type I pneumococci into the mastoid and the intratympanal and intracisternal areas had definite prophylactic value in the prevention of meningitis and septicemia.

3. Less definite prophylactic results were observed in rabbits inoculated intracisternally with virulent type II and type III pneumococci.

4. The simultaneous administration of ethylhydrocupreine hydrochloride by intravenous and intramuscular injection and of ethylhydrocupreine base internally, probably enhanced the prophylactic activity of the antipneumococcus serums.

5. In rabbits the intravenous and intramuscular injection of scarlet fever antitoxins had some prophylactic value in the prevention of meningitis and septicemia produced by the intracisternal inoculation of virulent hemolytic strains of streptococci.

6. It is suggested that the routine bacteriologic examination in chronic infections of the sinuses be made before operation and in mastoiditis at operation, to be followed by the prophylactic measures outlined in cases of unusual severity in which meningitis may occur.

MYOCLONIC MOVEMENTS OF THE LARYNX AND PHARYNX

A MANIFESTATION OF EPIDEMIC ENCEPHALITIS *

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Rhythmic involuntary jerking movements are sufficiently rare in the laryngeal and pharyngeal muscles to merit being recorded with whatever other associated phenomena may be present at the time. Accordingly, the clinical records in five such cases observed at the Mayo Clinic between 1926 and 1929, inclusive, are presented.

REPORT OF CASES

CASE 1.—A man, aged 51, came to the Mayo Clinic on Aug. 10, 1929, because of difficulty in breathing, spasmodic blinking of the eyelids and clumsiness in the use of the right arm. He had had a condition that was diagnosed as influenza on four occasions between 1918 and 1921. Following the last attack, he had noticed intermittent jerking movements of the right arm. About a year later, severe, sharp, shooting pains commenced in the same arm, with increasing stiffness and slowness of motion. In 1922, he became unable to follow his trade as a barber. In 1926, he occasionally noted a "clicking" sensation in the larynx, which seemed to obstruct breathing slightly, and one morning in 1927, he awoke to find it impossible to talk above a whisper. His voice returned in about two or three days, but since this episode it had never been quite normal. After talking for a short time there was a tendency to lose the voice, and at the same time there was a sensation of spasm in the larynx. After the appearance of these phenomena, the patient had some difficulty with inspiration. The air passages felt obstructed, and he was constrained to make grimaces while gasping for air. Rarely had he taken breath without difficulty, and occasionally also he had difficulty with expiration. His wife stated that he breathed normally during sleep. He had had no difficulty in swallowing. Since 1927, he had had an uncontrollable spasm of the eyelids which seemed to be worse when the respiratory functions were most affected. He had lost more than 6 pounds (2.7 Kg.) since the onset of the trouble.

The patient appeared to be somewhat undernourished and nervous. His voice was pitched low and was frequently interrupted while talking. He frequently assumed the attitude of a person on the point of sneezing. There were distinct loss of speed, weakness and clumsiness of the right arm, with interrupted single

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jerkings. There were frequently repeated blinking movements of the orbicularis oculi, which, however, were intermittent, irregular and not synchronous with other movements. Because of the difficulty in phonation, laryngoscopic examination was made. There was slight bilateral sensory and motor paralysis of the pharyngeal muscles, with complete loss of the pharyngeal reflexes. This apparently was not sufficient to cause difficulty in swallowing. As far as the vocal cords were concerned, adductor weakness was manifested by failure of the cords to approximate in phonation; this explained the frequent hoarseness. The outstanding feature, however, was the appearance, as seen through the laryngoscope, of constant, involuntary, rhythmic, bilateral adduction movements of the vocal cords. These occurred from about sixty to eighty times a minute and frequently interfered with inspiration by producing complete adduction before the range of inspiration was complete. There were also myoclonic twitchings of the facial muscles synchronous with those of the vocal cords. The pharyngeal muscles and those of the soft palate did not participate in the movements. A diagnosis of epidemic encephalitis with myoclonic movements of the larynx and pharynx and an early appearance of the parkinsonian syndrome was made.

The patient probably acquired epidemic encephalitis during one of his attacks of influenza. It is also probable that his trouble will increase, especially along the lines of rigidity and loss of speed in all muscular movements, with a more fully developed parkinsonian syndrome. The bizarre nature of the complaints, especially in reference to his breathing, at first suggested the diagnosis of hysteria. Examination of the larynx dispersed this suspicion, for the myoclonic movements were too rhythmic and out of range of voluntary effect to suggest a psychogenic origin. Further, they explained satisfactorily some of the patient's subjective complaints, although he had no suspicion of the existence of such movements.

Case 2.—A man, aged 54, came to the clinic on April 28, 1927, because of a mass in the right nostril. He had noticed this for about nine months, and it had been slowly growing larger. Seven years previously, he had had high fever and was stuporous for about five or six days; the condition was diagnosed as influenza. He had noticed difficulty in swallowing for several days thereafter, and did not regain his strength for about two months. Since then he had not noticed difficulties referable to the throat.

Examination disclosed a firm nodule about 3 mm. in diameter in the external anterior portion of the right vestibule of the nose. It was excised, and its base was cauterized. Microscopically, it proved to be an inflammatory process with subepithelial fibrosis. The pharynx and larynx were examined. A constant rhythmic jerking movement of the left pharyngeal wall could be seen. The superior constrictor muscles of the pharynx were pulled downward and to the left in this continuous myoclonic jerking at a rate of a little more than 60 a minute. The movement continued as long as the patient was observed and was present on each examination. The soft palate and vocal cords were not affected, the functions of the larynx and pharynx were not impaired, and the patient was totally unaware of this myoclonic phenomenon. A diagnosis of epidemic encephalitis, chronic phase, with myoclonic movements was made.

The patient had no complaints referable to the larynx and pharynx or the corresponding functions of talking, swallowing and breathing. The discovery of myoclonus was purely accidental. He probably acquired the epidemic encephalitis at the time of the original infection in 1920, or seven years previous to examination, when the disease was widespread in this country. The disease, however, had been mild, and the jerking movements had persisted as a chronic phase.

CASE 3.—A man, aged 45, came to the clinic on July 20, 1926, because of a burning sensation in his tongue. He had had a condition that was diagnosed as influenza in February, 1920. Three years before admission he had noticed an ulcer on the lower lip, which was removed surgically eighteen months later and diagnosed as carcinoma. He came to the clinic for special examination because he feared a recurrence.

Tissue removed at the operation elsewhere was available and was examined microscopically, but definite evidence of carcinoma could not be seen. Leukoplakia of the epithelium with subepithelial fibrosis and inflammatory reaction were present. During the examination of the pharynx and larynx, rhythmic jerking could be made out in the superior constrictor muscles of the right side of the pharynx and those of the right vocal cord, similar to the jerking in cases 1 and 2. The movements were quick, constant and rhythmic, with a rate from about 60 to 120 a minute, and they were present on four different occasions over a period of five months. The patient was not aware of them and had no complaints referable to swallowing, talking or breathing. The diagnosis suggested was epidemic encephalitis, probably contracted in February, 1920. The burning sensation in the tongue was regarded as psychogenic and due to fear of carcinoma.

In many particulars the condition in this case resembled that in case 2 in the more or less accidental discovery of myoclonic movements, the lack of awareness of their existence and the complete functional integrity of the parts involved. On the other hand, the movements were sufficiently striking and distinct to demand immediate attention on examination of the pharynx and larynx. Similarly, the movements were beyond the power of voluntary activity, and, like those in cases 1 and 2, could in no sense be regarded as psychogenic.

CASE 4.—A man, aged 45, came to the clinic on Oct. 10, 1927, because of clumsiness and tremor in the right arm and general weakness. In January, 1923, he had been ill for about three weeks with a condition that was diagnosed as influenza, and he had had a high temperature and delirium for the first few days. Following this, he had not regained his former strength. Two years before admission, he had noticed increasing clumsiness and stiffness in the right hand, so that finer movements, like writing, buttoning clothing and cutting food, had become increasingly difficult. A tremor appeared in the right arm when it was at rest, and all movements had become slower. For two years he had noticed gradually increasing fatigability and an increase in saliva, and for about six months his right foot had dragged in walking.

The patient was well developed. The face was masked, immobile and expressionless. All movements were slow and deliberate, and in walking the right arm was held stiffly at the side, being flexed at the elbow; there was constant tremor

in the hand when it was at rest. Speed was markedly diminished in all movements of the right arm and leg, and there was increased tonus in the muscles. Handwriting was performed slowly and with difficulty; the first few letters were large and well formed, but after a word or two the letters became small and almost illegible. He was unable to move the fingers of the right hand rapidly. The left arm and leg were also slow in movement but not as slow as the right. The voice was slow and toneless. Direct and laryngoscopic examinations of the throat disclosed constant, myoclonic jerking of the soft palate and pillars of the tonsil on the right side. The uvula was pulled up and to the right with each jerk. The superior constrictors of the pharynx also moved on the right side synchronous with the soft palate, but the vocal cords were not involved. There were about eighty movements a minute, which were regular and constant. The patient was unaware of these movements and had no trouble in swallowing or breathing.

The condition in this case was similar to that in cases 2 and 3. The significant feature of the case, however, was the typical parkinsonian syndrome. The patient's age and the history of the development of this characteristic syndrome left no doubt as to the presence of the chronic phase of epidemic encephalitis.

COMMENT

In these four cases essentially the same phenomenon, namely, myoclonic movements of the laryngeal and pharyngeal musculature, was presented. Careful examination of the affected organs was made in each case both by direct inspection and by endoscopy. In case 1, the myoclonic movements were confined to the vocal cords and had led to bizarre and puzzling symptoms which could be explained only by laryngoscopic examination. In cases 2 and 4 there was unilateral jerking of the superior part of the pharynx; the cords had escaped. In case 3, the vocal cord was involved on one side. In cases 2, 3 and 4, the patients were unaware of these movements and had no complaint referable to the throat. The patients in cases 2 and 3 did not have other symptoms of encephalitis beyond the myoclonic movements, and they came to the clinic with some other complaint, examination for which led to accidental discovery of the jerking. The patients in cases 1 and 4, however, had definite signs of more widespread involvement, and although one patient presented symptoms suggestive only of an early parkinsonian syndrome, the other presented a well defined syndrome, and for this he sought relief at the clinic. All four patients had previously had an infective febrile illness which had been diagnosed as influenza.

As far as the diagnosis in these four cases is concerned, since myoclonic movements are so common in epidemic encephalitis and so rare in other diseases, it was natural to consider that disease first. Evidence

was present in cases 1 and 4 to corroborate this diagnosis, and in cases 2 and 3 it was reasonable to assume that the same condition caused the movements. Generally speaking, there is no known disease of the central and peripheral nervous system that is capable of producing such inclusive symptoms as epidemic encephalitis;[1] thus there is an ample possibility of error in including all sorts of strange and unfamiliar symptoms in this category. Accordingly, certain criteria must be established and a reasonably comprehensive differential diagnosis must be kept in mind. Such criteria might include the presence of a parkinsonian syndrome, as well as a history of an initial infective episode with or without paralysis of the cranial nerves.

Owing to the possible failure to realize that there is a chronic form of epidemic encephalitis, difficulties have appeared in the recognition of the minor manifestations of the disease. At the outset the acute forms were soon recognized, but it took several years to realize that an acute attack might be followed months or years later by progressive crippling; that, in fact, the acute form might become chronic. At first the latter manifestations were wrongly labeled "postencephalitic sequelae," and so forth, but now it is recognized that after a period of months or years, during which the patient is apparently free from symptoms, symptoms may again appear, indicating the persistence of the disease. This remission between the original acute attack and the later slowly progressive forms occurs in other diseases of the nervous system, notably multiple sclerosis and syphilis of the central nervous system. To carry the analogy further, the initial episode may be so mild as even to be forgotten, or it may be covered under the diagnosis of "influenza" or even "common cold." There may be successive waves of infection, as in case 1, and sooner or later the chronic stage makes its appearance and slowly progresses either to a point at which it remains stationary or to complete helplessness of the patient. At any time during this period of months or years after the original infection, peculiar phenomena, such as the phenomenon considered here, may appear even when the patient seems to be normal. They may disappear after a time, to be followed later by other manifestations, which change in character from time to time. In the four cases cited, the disease was in the chronic relapsing phase, and the patients' futures must of necessity be uncertain and full of morbid possibilities.

Epidemic encephalitis may produce paralytic phenomena, with suspension of movement, or kinetic phenomena, with exaggeration of

1. Riley, H. A.: Epidemic Encephalitis, Arch. Neurol. & Psychiat. **24**:574 (Sept.) 1930.

movement, in any part of the musculature of the body controlled by the central nervous system. There may be a combination of paralysis and disorders of movement. The movements may be universally distributed in the body and involve many muscles or they may be localized to a small area, as in these four cases. In epidemic encephalitis disorders of movement may be divided into general and local convulsions, chorea, athetosis, dystonia, tic, spasm, tremor, myoclonia and, finally, movements so complex that they can hardly be described.

The jerking characteristic of myoclonia is sudden, quick and like that produced in a muscle by intermittent faradic shocks.[2] The muscle relaxes quickly but contracts again rapidly, so that continuous twitching is present that varies in speed in different cases. These movements may occur during the acute phase of epidemic encephalitis [3] or may appear as chronic manifestations months or years afterward, as occurred in the four cases reported. Myoclonic movements did not occur commonly in the epidemic of 1920 and 1921; they were more common in 1922 and 1923, and ever since they have continued to appear either as isolated signs or as part of a chronic syndrome of encephalitis.

The jerkings may be universal and may involve most of the voluntary muscles of the body; these phenomena were more common in the early years of the epidemic. The cases were characterized by an acute onset, with sharp, shooting pains in the trunk and extremities, at first local but later becoming generalized. The pains were soon followed by muscular jerks, waves and twitchings, which were often rhythmic and persisted for weeks or months. Apart from these generalized myoclonic disturbances seen early in the epidemic and during more recent years, less severe cases were observed and recorded. Moreover, the jerkings were not so widespread and were more chronic. The abdominal muscles are particularly vulnerable and may be involved, unilaterally or bilaterally. The legs or an arm and a leg on one side may be affected. Still more locally, the jerkings may involve a single limb, a group of muscles, one muscle or even a segment of a muscle. They are frequently present during sleep; occasionally they are absent. They may change from one part to another, and different groups of muscles, even with different rates of jerking, may be involved in the

2. Stewart, J. P.: The Diagnosis of Nervous Diseases, London, E. Arnold & Company, 1924, p. 119.

3. Hunt, J. R.: Acute Infectious Myoclonus Multiplex and Epidemic Myoclonus Multiplex (Epidemic Encephalitis), J. A. M. A. **75**:713 (Sept. 11) 1920. Reimold, Werner: Ueber die myoklonische Form der Encephalitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:21, 1925. Sicard, J. A.: L'encéphalite myoclonique, Presse méd. **28**:213 (April 14) 1920.

same patient. Krebs [4] found the jerking to be diminished with muscular exertion, whereas mental fatigue and emotional excitement exaggerated the movements.

As has been mentioned, the abdominal muscles are particularly prone to attacks, but so is the diaphragm, and recurrent epidemics of hiccup due to recurrent myoclonia of that muscle have been recorded.[5] The tongue, face, jaws, larynx and pharynx have been affected with about equal frequency.[6]

Association of these myoclonic movements with some other syndrome of epidemic encephalitis has been helpful in establishing the diagnosis. Two of the patients described in these four cases had Parkinson's syndrome, which has been well recognized as highly suggestive of epidemic encephalitis. Occasionally with the more simple myoclonic jerkings there are more complicated movements, in themselves suggesting epidemic encephalitis. Mention may be made of the "pill-rolling tremor" of the parkinsonian syndrome, spasms of the orbicularis muscle and the extraordinary attacks of forced conjugate upward movements of the eyeballs. Frequently, myoclonic movements disturb the respiratory rhythm or depth when they affect the intercostal muscles, the diaphragm, the accessory muscles of respiration and, as in case 1, the muscles moving the vocal cords. Turner and Critchley [7] classified these respiratory disorders into: (1) disorders of the respiratory rate (tachypnea, bradypnea); (2) disorders of the respiratory rhythm (Cheyne-Stokes respirations, breath-holding spells, sighs, forced or noisy respiration and inversion of the inspiration-expiration rate), and (3) respiratory tics (yawning, hiccup, spasmodic cough, sniffing, sobbing and paroxysmal screaming). Further descriptions of these curious clinical phenomena have been recorded.[8] Myoclonic

4. Krebs, M. E.: Des caractères cliniques des secousses musculaires au cours de l'encéphalite épidémique: Rapports des myoclonies observées actuellement avec quelques syndromes antérieurement connus; les mouvements spasmodiques rythmés dans l'encéphalite, Bull. et mém. Soc. méd. d. hôp. de Paris **1**:522 (March) 1922.

5. Brain, W. R.: Epidemic Hiccup and Encephalitis Lethargica: Report of a Case of Epidemic Encephalitis Showing Lethargy, Cranial Nerve Palsies, Myoclonus and Hiccup, Brit. M. J. **2**:856 (Nov. 10) 1923.

6. Dahl, J. F.: Case of Dysarthria with Myoclonia, Med. rev. Bergen **46**: 49 (Feb.) 1929. Jelinek, D., and Sachs, A.: Clinical Study of the Pathology of Rhythmic Myoclonia of the Palate and Larynx, Časop. lék. česk. **69**:64 (Jan. 10) 1930.

7. Turner, W. A., and Critchley, MacDonald: Respiratory Disorders in Epidemic Encephalitis, Brain **48**:72 (March) 1925.

8. Parker, H. L.: Disturbances of the Respiratory Rhythm in Children: A Sequela to Epidemic Encephalitis, Arch. Neurol. & Psychiat. **8**:630 (Dec.) 1922; Epidemic Encephalitis: Respiratory Syndrome, M. Clin. North America **13**:1347 (May) 1930.

movements of the diaphragm without hiccup but with simple tachypnea have been visualized by the fluoroscope and relief of the condition has been obtained by cutting or freezing one or both phrenic nerves.[9] In epidemic encephalitis, however, most respiratory difficulties are vastly more complicated than simple myoclonic movements of the diaphragm, intercostal muscles or vocal cords.

Under the caption, palatolaryngeal nystagmus, Wilson[10] described the syndrome in a case of hypertension and arteriosclerosis. A recent apoplectic insult had been attended with right-sided convulsions. Oscillation of the palate at the rate of 144 a minute was noted. There were also horizontal movements of the left superior constrictor and left posterior faucial pillar. With a laryngoscope, well marked twitchings of the left arytenoid and left vocal cord could be seen synchronous with those of the palate. In discussion of this case, Critchley[11] stated that although in a given case it was hard to correlate the pathologic and clinical observations, this variety of movement is not uncommon in cerebrovascular disease. Such movements had been seen in focal softenings of the dentate nucleus or in the pons. Critchley also mentioned tumor of the vermis cerebelli as a possibility, and corroboration of this can be given by the following brief record of a case from our files.

Case 5.—A boy, aged 11, came to the clinic on Oct. 29, 1925, because of unsteadiness of gait, vomiting and headaches. Seven months before admission, double vision had developed, and five months before, unsteadiness in gait, which had progressed. About the same time he began to stagger in walking and severe, intermittent headaches with projectile vomiting appeared. One month previous to admission, it had been noticed that the right side of his face was paralyzed and that he used his hands clumsily. All symptoms were becoming progressively worse.

The boy appeared to be undernourished. Paralysis of the right side of the face was obvious, as were stagger in walking, universal ataxia and clumsy use of the right side of the body. Speech was indistinct, and there was wide nystagmus in all directions. Ophthalmoscopic examination disclosed papilledema of both optic disks of from 1 to 2 diopters. Rapid twitching movement of the right half of the soft palate and upper part of the pharynx was noted. The movements were about 140 a minute and continuously rhythmic. There was no paralysis of the palate, larynx or pharynx. Craniotomy was performed through a suboccipital approach,

9. Dowman, C. E.: Relief of Diaphragmatic Tic, Following Encephalitis, by Section of the Phrenic Nerve, J. A. M. A. **88**:95 (Jan. 8) 1927. Gamble, C. J.; Pepper, O. H. P., and Muller, G. P.: Postencephalitic Tic of the Diaphragm: Pulmonary Overventilation and Relief by Blockade of the Phrenic Nerve, J. A. M. A. **85**:1485 (Nov. 7) 1925.

10. Wilson, S. A. K.: Palato-Laryngeal Nystagmus, Brain **51**:119 (March) 1928.

11. Critchley, MacDonald: Discussion, Brain **51**:119 (March) 1928.

and a soft, apparently rapidly growing tumor was found in the right cerebellar hemisphere, extending into the vermis cerebelli and partially filling the fourth ventricle.

Small hemorrhages in the brain can produce a syndrome similar to that just cited. LaSalle and Bédard [12] described the case of a man, aged 28, who complained of severe vertigo that had suddenly appeared following his departure from a compressed air caisson. Hearing had diminished in the right ear, and there was a positive Romberg sign. There was rhythmic jerking of the soft palate and the suprahyoid muscles. These movements were rhythmic and recurred about 60 times a minute. The authors concluded that the patient had suffered from small focal hemorrhages in the region of the upper medulla and pons affecting the vestibular nuclei and the intramedullary fibers of the glossopharyngeal nerve just distal to their origin from the nucleus. Accordingly, it is possible to find similar rhythmic spontaneous movements in the soft palate, pharynx or larynx in the course of encephalitis, encephalomalacia, hemorrhages and tumors. In our experience, encephalitis seems to be by far the most common cause of myoclonic movements. The extraordinary case described by Stoll [13] might be mentioned in the category of rhythmic movements of unknown cause. This was of a mentally deficient man, aged 30, who had complained of a loud, ticking noise in both ears for twelve years. With the aid of a nasopharyngoscope, rhythmic contraction of the salpingopalatinus muscles of the eustachian tube was observed. With each contraction, the orifice of the eustachian tube could be seen to open, and with each relaxation, to close. This made a noise identical to that when moistened lips are separated. It could be heard by the observer 50 cm. from the patient's left ear and recurred about 132 times a minute. A similar but fainter noise recurring 108 times a minute could be heard with the observer's ear in contact with the patient's right ear. A similar case has been reported by Houser.[14]

Similar movements of the larynx and pharynx have been described, but need be mentioned only briefly in the differential diagnosis. In both acute and chronic forms of chorea, irregular motions occur in the face, tongue and pharynx as well as in the respiratory muscles, pro-

12. LaSalle and Bédard: Nystagmus du voile: syndrome myoclonique de la calotte protubérantielle, Union méd. du Canada **56**:627 (Nov.) 1927.

13. Stoll, H. F.: Tic of the Salpingopalatinus Muscle Causing Sound Audible to the Bystander, J. A. M. A. **95**:796 (Sept. 13) 1930.

14. Houser, K. M.: Clonic Spasm of the Muscles of the Eustachian Tubes, Arch. Otolaryng. **7**:289 (March) 1928.

ducing grunting noises. Lhermitte and McAlpine[15] described chronic movements of the face, palate and larynx. Grabower[16] described a tremulous movement of the vocal cords in multiple sclerosis, tabes and tumors of the frontal lobe. Greene[17] also has added to observations on the vocal cords in tabes. In multiple sclerosis there is either a coarse active tremor or jerking movements occurring usually in phonation and sometimes in respiration. Prolonged sound is impossible, and the voice quickly becomes falsetto. Similarly, ataxic movements of the vocal cords occur in the presence of lesions of the cerebellum, and have been described by Bender.[18] In true myoclonus of the larynx or pharynx, a psychogenic cause cannot be entertained. In the first place the movements are completely outside the domain of purposive activity, and the patient is frequently unconscious of their presence. In the second place, they are too regular, rhythmic and relatively simple in their mechanism to be confounded with the more complex, ever changing tics, mannerisms and habit spasms of nervous and emotionally unstable persons.

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15. Lhermitte, Jean; and McAlpine, Douglas: A Clinical and Pathological Résumé of Combined Disease of the Pyramidal and Extrapyramidal Systems, with Especial Reference to a New Syndrome, Brain **49**:157 (June) 1926.

16. Grabower: Die diagnostische Bedeutung der Kehlkopflähmung, Berl. klin. Wchnschr. **1**:664 (April 10) 1911.

17. Greene, D. C., Jr.: A Study of the Larynx in Tabes, Boston M. & S. J. **154**:97 (Jan. 25) 1906.

18. Bender, Lauretta: Cerebellar Control of the Vocal Cords, Arch. Neurol. & Psychiat. **19**:796 (May) 1928.

FATAL HEMORRHAGE RESULTING FROM THE EXTENSION OF A RETROPHARYNGEAL ABSCESS TO THE DEEP VESSELS OF THE NECK*

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Erosion and perforation of the important blood vessels of the neck, as the result of extension to their coats of suppuration from surrounding tissues, are comparatively rare. A limited number of cases of fatal or very serious hemorrhage occurring in this way from the extension of deep cervical abscesses, especially those following scarlatina, have been recorded. In the present report I shall refer only to fatal or serious hemorrhage occurring as a complication of retropharyngeal abscess by the involvement of the deep cervical vessels, especially the internal carotid artery and the jugular vein.

Although the number of cases reported is limited, the possibility of hemorrhage should always be borne in mind, because in such an event nothing but the most prompt intervention will save the patient's life. Moreover, a consideration of the clinical cases as well as of the anatomic conditions, which might almost be said to predispose to the possiblity of such a catastrophe, should warn one to take prophylactic measures to avoid it. The spreading of infection from a retropharyngeal abscess may be insidious and rapid, and the clinical symptoms of the involvement of important vessels may be entirely absent. Early incision and evacuation of every such abscess are therefore imperative if one is to guard against the possibility of a fatal result.

Before reviewing the literature on the subject or referring to the anatomy of the retropharyngeal region and the probable causes of hemorrhage in the presence of a retropharyngeal abscess, I shall give a brief history of a case that I recently observed, in which a retropharyngeal abscess was complicated by a sudden, spontaneous and fatal hemorrhage that probably originated from the internal carotid artery.

REPORT OF CASE

A boy, aged 3 years, had been ill for five days with sore throat, slight earache on both sides and general malaise. He was treated by the family physician. The temperature ranged between 101 and 103 F. Two days later, a swelling appeared on the right side of the neck below the angle of the jaw; this swelling became progressively larger.

* Submitted for publication, Feb. 3, 1931.

The past history was irrelevant, except for breathing through the mouth and recurrent colds and sore throat.

On careful examination, the child did not look very ill, but was quite restless. He sat up in bed, talked with a slightly muffled voice, took nourishment well and complained of pain in the right side of the neck. Occasionally, there was pain in the ears. At this time his temperature was 102 F. Physical examination revealed a slight injection of the drum membranes of both ears, especially about the hammer handle, but there was neither bulging nor obliteration of landmarks. The nasal mucosa was considerably congested without visible pus; the throat looked red; the tonsils were large, almost meeting in the midline, and the crypts were filled with mucopurulent material and débris of food. The oropharynx was injected, and a small swelling, the size and shape of an almond, was found hidden behind the right posterior pillar in the region of the lateral fold; this swelling was firm and smooth, extended to the lower level of the tonsil and did not interfere with the patient's swallowing. There was another swelling, the size of a hen's egg on the right side of the neck, which was composed of groups of glands that had not yet broken down. There were other enlarged cervical glands on both sides of the neck.

A right-sided retropharyngeal abscess was suspected. Incision was advised, but was refused by the parents.

The day following the examination it was reported that the child was better. On the morning of the third day, a little blood appeared in the sputum. The parents did not consider this of consequence and did not call me. The fourth day the child vomited old blood, coughed and spat bloody sputum; there were increased pallor and restlessness. I advised taking him to the hospital at once. While on the way to the hospital, he had a sudden profuse and propulsive hemorrhage and died from exsanguination before reaching the hospital. Autopsy was not allowed, but external examination of the body post mortem showed extreme exsanguination; the ears showed nothing abnormal; the nostrils and mouth contained clotted blood. On the right side of the throat behind the right tonsil and in the middle of the swelling that had been seen at the time of the first examination, there was a crater-like cavity filled with clotted blood and pus. A probe that was introduced into the fistula from the bottom of the crater led in the direction of the large vessels of the neck. The immediate course of the bleeding, however, could not be ascertained without dissection, but the presence of bright red blood, the suddenness of the hemorrhage and its profuseness speak most probably for the involvement of the carotid artery and possibly the internal branch, as this was nearest the location of the abscess. On inspection, evidence of vascular anomalies was not found.

REVIEW OF THE LITERATURE

A review of the literature disclosed reports of twenty-two other cases of fatal or serious hemorrhage as a complication of retropharyngeal abscess. These cases are shown in the accompanying table.

The summary shows that in cases of retropharyngeal abscess, hemorrhage is more common from the internal carotid than from any other vessel; retropharyngeal abscess and hemorrhage are more common in young patients than in adults, and hemorrhage is more frequent in males than in females; hemorrhage is usually secondary, following the opening of the abscess; the left side is more often the site of hemorrhage than the right. Ligation of the common carotid appears to be

the only treatment that assures recovery when hemorrhage takes place, since three of the four recoveries followed this procedure.

ANATOMY OF THE RETROPHARYNGEAL REGION

The mode in which the internal carotid artery or internal jugular vein may become ulcerated and perforated during the evolution of a retropharyngeal abscess will be easily understood from a consideration of the anatomy of the region.

Wishart,[1] in considering the anatomy of the posterior wall of the pharynx, reproduced three plates from Eycleshymer and Shoemaker's "Cross Section Anatomy." The first plate, taken at the level of the hard palate, shows a thin strip of muscle directly behind the mucosa of the pharynx; behind this is a thin layer of connective tissue represent-

Cases of Fatal or Serious Hemorrhage as a Complication of Retropharyngeal Abscess Found in the Literature

Total number of cases	23	Thrombosis of jugular vein	1
Number of deaths	19	Nature of hemorrhage:	
Recoveries	4	Spontaneous	6
(*a*) Following ligation of common carotid	3	Secondary	12
(*b*) Without ligation	1	Traumatic	3
Vessel from which hemorrhage occurred:		Not stated	2
Internal carotid	10	Age of youngest patient	5 weeks
Common carotid	2	Age of oldest patient	25 years
Common carotid, probably	1		
External carotid	1	Where started:	
Internal jugular	1	Left side	13
Collaterals	1	Right side	5
Not known	7	Sex:	
Total	23	Males	10
		Females	2

ing the retropharyngeal space at this level. This connective tissue is bounded posteriorly by two muscles, the recti capitis anticus, major and minor. At the sides, the retropharyngeal space is continuous with the pharyngomaxillary fossa. In the plate showing the cross-section at the level of the tongue, the retropharyngeal space is less evident, but the pharyngomaxillary fossa is better defined and is here divided, as was first demonstrated by Zuckerland, into two chambers, the anterior and the posterior. The posterior chamber contains in its hindmost part the internal carotid artery, the internal jugular vein and their accompanying nerves. This chamber and its contents deserve particular attention in cases of hemorrhage arising from abscesses here.

Mosher [2] said that the pharyngomaxillary fossa is roughly funnel-shaped, the apex being below. The carotid sheath emerges at the apex of the fossa and continues the fossa through the neck to the thorax.

1. Wishart, E. S.: Canad. M. A. J. **13**:635, 1923.
2. Mosher, H. P.: Tr. Am. Laryng. Rhin. & Otol. Soc., 1920, p. 327.

Wishart presented a schematic representation of the pharyngomaxillary fossa, showing that the large vessels lie in the center of the retropharyngeal space and are thus prominently exposed to purulent collections in this region.

Anatomically, also, the retropharyngeal space is most admirably located as a focus of infection. In the first place, it theoretically extends upward to the septum nasi. Practically, however, according to Wishart, it extends only a short distance, as it is limited by the subpharyngeal connective tissue which binds the mucosa firmly to the anterior face of the basilar process of the occipital bone. There is practically no downward limit to the possible extension of the retropharyngeal space; hence, complication of the mediastinum and larynx is not uncommon. In his book St. Clair Thomson [3] reproduced a section through the cervical vertebrae, pharynx and adjacent parts, showing the cavity of a retropharyngeal abscess. The abscess extended from the level of the basioccipital bone to the level of the fifth cervical vertebrae below.

The boundaries of the fossa show that it is open to infection from all sides. In retropharyngeal abscesses the infection may proceed from the nose, the throat or the ear, and this spreading of infection is particularly favored by the lymphatic drainage. It will be of importance, therefore, to study the disposition of the lymphatics.

The superficial cervical lymph nodes lie along the external jugular vein; the deep cervical nodes lie on the internal jugular and are often adherent to it. All of the superficial lymph nodes drain into the deep ones.

According to Wishart, the retropharyngeal lymph nodes are arranged in four groups, two on each side of the midline of the pharynx. The lateral group is found median to the internal carotid and is described as more constant, except in children, than the median group, which lies in the retropharyngeal space closer to the midline. Both groups send efferent vessels to the internal jugular group of the posterior deep cervical chain. Infection coming from the nose or pharynx easily breaks down the lymph nodes in the pharyngomaxillary fossa and forms a retropharyngeal abscess. The formation of such abscesses is much more common in infants and children than in adults, owing to the greater prevalence of infectious diseases in childhood and to the fact that lymph nodes are much more numerous in the retropharyngeal space of the child than in that of the adult. A fuller description of the lymphatics of the retropharyngeal region and a review of the literature are given in an article by Frank,[4] which also shows their connections with the nose and ear.

3. Thomson, St. Clair: Diseases of Nose and Throat, New York, D. Appleton & Company, 1916, p. 638.

4. Frank, Ira: Retropharyngeal Abscess, J. A. M. A. **77**:517 (Aug. 13) 1921.

VASCULAR INVOLVEMENT IN RETROPHARYNGEAL ABSCESS

Having outlined the anatomic details concerned with the matter under discussion, I shall proceed to a consideration of the question of the direct attack of suppuration on the vascular tunics in the presence of retropharyngeal and other deep-lying cervical abscesses. For a long time it was orthodox to believe that the tunics of vessels were capable of resisting infection. It is certain, indeed, that nature has here constituted a strong barrier to the spread of infection, and blood vessels are usually the last to yield in the case of an abscess or sloughing infection. This relative immunity or resistance to infection may be accounted for by several factors, all of which serve to give the vessels an added protection. One factor is the presence of the vasa vasorum. The larger arteries and veins are surrounded by a rich network of perivascular lymphatics and minute nourishing vessels which, so long as they themselves are free from infection, contribute in no small degree to the fighting capacity of the larger vessels against invading microorganisms.

The character of the vascular coats is another factor. The tunica media is a fairly thick fibromuscular coat. The external tunic or adventitia is a well knit, firm coat, composed of interlacing and elastic fibers, while the inner tunica is covered by endothelium that is probably endowed with a special phagocytic quality. The presence of a fascial sheath, like the carotid sheath, which envelops the larger vessels, serves as an insulator and as an additional safeguard against external attack.

Nature, however, has provided additional safeguards. The reaction of the blood vessels to infection is of such specific character as to shield them from sudden rupture and ensuing hemorrhage. Usually when a vein is attacked, there is a regular pathologic sequence of periphlebitis, phlebitis and thrombus formation, either mural or as a central plug, resulting in embolic dispersion with sepsis or leading to an occlusion of the vessel and its entire obliteration.

In an artery the blood stream is too swift and the pulse pressure too great to allow easy clotting. The force of this pressure, however, acting against a weakened spot in the arterial wall will tend to produce an aneurysm or sacculation. In either case, the pathologic process is relatively slow and of such character as to prevent sudden rupture and severe hemorrhage.

ETIOLOGIC CONSIDERATIONS

From the foregoing remarks the etiologic factors concerned with a hemorrhage from the deep vessels of the neck during the evolution of a retropharyngeal abscess appear to be evident enough. They are: (1) the involvement of the superficial or deep lymphatic glands by any

infective process, (2) the proximity or actual contiguity of either the internal carotid artery or the internal jugular vein to the deep lymphatic glands, (3) infection, either acute or chronic, from the nose, throat or ear, which may reach the retropharyngeal space by any route, (4) the peculiar virulence of certain bacteria that may have a predilection for attack on the walls of the vessel, (5) the presence of vascular anomalies and (6) the presence of vascular disease.

In none of the cases that I have collected is mention made of vascular anomaly or a vascular disease. Nor has there been, so far as I can find, any mention of special bacterial action on the walls of the vessels. I mention these etiologic factors because they seem possible rather than supported by reported facts. In most of the reported cases, however, extensive pathologic examinations were not made.

The most important etiologic factor is the anatomic disposition of the blood vessels in the presence of a retropharyngeal abscess. The vessel is directly exposed to the spread of infection.

Liston,[5] in 1843, was probably the first, despite the strong opposition of his Medical Society, to show that a chronic abscess of the cervical region could directly communicate with the carotid artery. A number of similar cases were soon published and proved that chronic abscesses could expose and ulcerate vessels. In 1871, Gross reported a personal case and collected eleven others in which there was fatal hemorrhage from perforation of the jugular vein due to diffuse or gangrenous cellulitis following scarlatina. Gross also collected five cases of hemorrhage into the common carotid artery and five of hemorrhage into the internal carotid artery, with apparently the same etiology. In 1889, Fischer [6] greatly increased these statistics. He collected forty-two cases of arterial hemorrhage following scarlatinal abscesses in the cervical region, of which twenty-eight had a fatal termination. In addition, there were twenty-eight collected cases of venous hemorrhage, including Gross' cases, twenty-three of which clearly involved the jugular vein, with twenty-one deaths. There was also six cases of the same kind of abscess in which both the carotid artery and the jugular vein were simultaneously attacked; all six of these cases ended fatally. Fischer mentioned the fact that retropharyngeal abscess is not an uncommon complication of scarlatinal angina, but he did not include any direct case of retropharyngeal abscess in his list, except that of Szekeres, which is included in my list of cases.

In all of these cases, the hemorrhage was from an eroded vessel into the cavity of a closed abscess or into the cavity several days after the contents had been evacuated. This usually occurred in children

5. Liston, R.: Brit. & Foreign M. Rev. **15**:155, 1843.
6. Fischer, H.: Arch. f. klin. Chir. **18**:367, 1899.

convalescing from scarlatina, but a few were cases of scrofulous abscesses in adults. The pressure of an abscess against the wall of a vessel may check an impulsive hemorrhage from the vessel, or the abscess cavity may be filled with blood and clot; the hemorrhage occurs through the mouth and nose only when the pressure has been relieved by evacuation of the abscess.

The presence of an anomaly may facilitate the erosion of vessels. In the case of the internal carotid there are well known anomalies in the pharyngeal region, as has been shown by Kelly[7] and Fischer.[8] These writers show that the internal carotid originates from the third aortic arch and dorsal aorta. At the junction of these two parts a pronounced bend is formed, either forward or medially. As embryonal development proceeds, the artery is straightened out as the heart recedes into the thorax. If the straightening is incomplete, the embryonic bend persists, producing undulation, angulation and tortuosity of the vessel. Tortuosity of the internal carotid may be coronal or sagittal. When it is coronal, it is liable to injury during operations involving the posterior wall of the pharynx, such as removal of the adenoids or incision of a retropharyngeal abscess. In the sagittal plane, the vessel is more likely to be injured during operations on the tonsils or incision of peritonsillar abscesses. Such an anomaly appears more commonly in children than in adults and plays an important rôle in the erosion of the vessel by a retropharyngeal abscess.

In the majority of the reported cases, the hemorrhage proceeded from the internal carotid artery. It proceeded from the internal jugular vein in only one case. From an anatomic point of view, it would seem that the jugular vein would be the vessel more frequently involved because of its close association with the lymphatics. This may be accounted for by the fact that an occlusion of the vessel is more likely to occur than a hemorrhage. Mosher expressed the belief that the most important structure in the pharyngomaxillary fossa, from the standpoint of infection, is the internal jugular vein. As stated before, the veins are strengthened against ulceration by fibrinous deposits, while the arterial walls are not. Thrombosis of the internal jugular vein, therefore, should always be considered as a possible complication of a retropharyngeal or other deep-lying cervical abscess, as should hemorrhage from this vessel or from the internal carotid artery. The complication may occur as sinus thrombosis or jugular thrombosis in the presence of infection of the middle or the internal ear. Mosher reported a personal case of this kind and referred to others; he hinted that infections of the lateral sinus or of the internal jugular vein do not always come from the mastoid, but may come through the tonsil or elsewhere.

7. Kelly, A. Brown: J. Laryng. & Otol. **40**:15, 1925.
8. Fischer, A. G. T.: Lancet **2**:128 (July 17) 1915.

PATHOLOGY

The pathologic conditions found at autopsy in cases of hemorrhage associated with retropharyngeal abscess show the breaking down of the lymphatic glands with more or less extensive abscess involving the pharyngomaxillary space, and extension of the suppuration to the walls of the vessels with phlebitis or arteritis and hemorrhage. Where the vessel is perforated, there may be a limited necrosis of its wall, owing, in the venous cases, to the cutting off of the vascular supply or, in the case of pressure by the abscess, to the withdrawal of the contents, or there may be progressive inflammatory changes. The abscess cavity is usually filled with clotted blood, and its anterior wall, which forms the posterior wall of the pharynx, is ruptured where the hemorrhage escapes into the mouth.

PROPHYLAXIS AND TREATMENT

In all children with acute sore throat and large projecting tonsils, a thorough search should be made behind the posterior pillars for the presence of a retropharyngeal abscess. A retropharyngeal abscess is always a dangerous condition, and whenever such a lesion is suspected or verified, temporizing is not justifiable. The abscess should be incised at the earliest opportunity; one should keep toward the median line as far as possible and use a blunt instrument. Aspiration or suction may be advisable in some cases, and every effort should be made to evacuate pus, thus checking the spread of the infection. Mosher expressed the opinion that in every case of peritonsillar or retropharyngeal abscess, active or quiescent, that is associated with chills or a septic temperature, there is probably thrombosis of the internal jugular vein.

The slightest sign of hemorrhage, even if there is only a slight amount of blood in the sputum or vomitus, associated with a retropharyngeal abscess is an indication of danger and calls for immediate action. Such a symptom may be followed, as in many of the cases reported, by a sudden, profuse and fatal hemorrhage. In such a hemorrhage, packing the cavity has little effect; compression of the vessels against the vertebrae is only a temporary expedient, and attempts at electrocoagulation are impracticable and dangerous. Galloway thinks that a sudden and profuse hemorrhage associated with a retropharyngeal abscess is an indication for immediate ligation of the internal or the common carotid. Study of the statistics shows that this is the only effective treatment, and that when executed in time usually saves the patient's life. Of course, it may not be possible to localize the hemorrhage to the internal carotid, and ligation of the corresponding common carotid is indicated. The immediate mortality from the operation is small, and ultimate complications occur in only about 25 per cent of

the cases. I think that in the case reported, ligation would have saved the patient's life.

The question of leaving a drainage tube in situ in cases of incised retropharyngeal abscesses deserves special consideration. The fatal results in Beyme's and other cases, in which erosion of the involved walls of the vessels was evidently due to a drain left in place for several days, suggest that this type of drainage would best be omitted.

A STUDY OF TINNITUS*

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Tinnitus is a pathologic symptom. The quantitative study of it seems never to have been carried to a point of utility in diagnosis, or of fitting in with any quantitative theory of hearing. Tinnitus is described as being broadly of two kinds: the external, which originates in the middle ear and is due to mechanical causes resulting in actual sound such as the spasmodic contraction of the tensor muscles, and the internal tinnitus, which has associated with it no sound, but originates in the labyrinth as a result of some disturbance of the metabolic equilibrium of the nerves or their associated apparatus. I am under the impression that the presence of tinnitus of either kind generally indicates an active or progressive lesion, and that a cessation of it, perhaps as a result of treatment, is an indication that the progress of the degeneration or atrophy of tissue has been arrested. My own experience is that people entirely without tinnitus are extremely rare, if such cases exist at all. People are usually not aware of tinnitus until it becomes so loud as to interfere considerably with perception of the weaker sounds. In the belief that quantitative measurement of tinnitus is of utility, both theoretically and in diagnosis, and in the hope that a description of some measurements with the conclusions arrived at will interest others to the point of making similar studies, I shall give a brief account of some measurements made on one of my own ears about three years ago. These measurements constitute a study of the "abnormal sensation areas."[1]

Chart 1 is an audiogram of the ear in question. It shows an abnormal loss of acuity in the range of from 3,500 to 10,000 cycles per second. External examination of the ear indicated that the tympanum was in good condition, and this, added to the fact that the frequencies affected are high, suggests strongly that this is a disease at the terminals of the nerves along the proximal one third and one fourth of the organ of Corti, except at the very end. Calculated on the basis of an average ear, the distal end of this affected region is at about the 8 mm. point;[2] but, judging from individual variations in ears, this point might be anywhere from 6 to 12 mm. from the round window.

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* Submitted for publication, Dec. 8, 1930.

1. Wegel, R. L.: Nat. Acad. Sc., 1921; Proc. Triological Soc., 1921.

2. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Tr. Am. Otol. Soc. **18**:262, 1928.

The tinnitus was not very loud, but it was sustained or continuous and of high pitch and sounded very much, though not exactly, like a tone of 3,600 or 3,700 cycles per second as indicated. If the "internal" hypothesis is correct, this suggests that the nerve terminals or their corresponding ganglion cells at and about the nominal 8 mm. point are being continuously stimulated by an excess of toxic ingredients. This deduction, together with the collateral circumstance that chronic nerve deafness with very few exceptions attacks the end-organ in this region,[2] is the principal basis for the speculative conclusion that the abundance of supply of nourishment by the blood is more precarious here than elsewhere. It might be objected that there is no conclusive evidence from histology of the blood supply to bear out this point, but the functional evidence noted is submitted as being sufficient ground for at least suspecting something of this nature. In a few cases of trauma approxi-

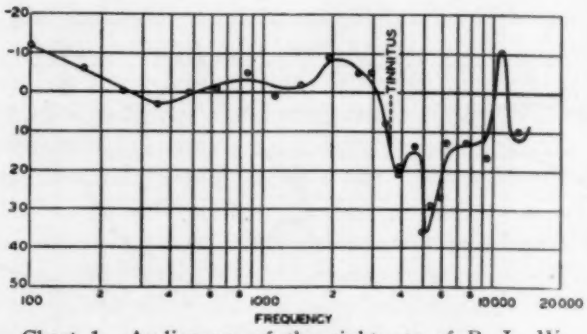

Chart 1.—Audiogram of the right ear of R. L. W.

mately the same region is affected, so it is possible that all such cases result from trauma, but this is rather unlikely.

Chart 2 represents the threshold and "masking" curves of the tinnitus. These data were repeated on the same ear on five different days and with different telephone receivers, each time with the same results. The top curve is the threshold, but was not plotted with respect to normal. Its shape, therefore, is of no importance in this consideration, as it depends to a certain extent on the efficiency of the telephone receiver at different frequencies. The receiver with which data of chart 2 were taken is one that is capable of delivering sound of very high purity at high intensities as well as low.

Measurements of the threshold were made with extreme care on two occasions, and it was found to fluctuate by 3 sensation units in synchronism with the pulse in the range of frequencies of from 2,000 to 4,000 cycles. This suggests that the vas spirale in its periodic pulsations causes these fluctuations of sensitivity, which correspond to fluctuations in actual amplitudes of vibration of about 40 per cent. No fluctuations

in pitch were perceptible, so it is concluded, from the discrimination data on the pitch, that these were less than 0.3 per cent, corresponding to a shift of the vibrating spot along the membrane of less than about 1/60 mm. No fluctuations in either pitch or intensity were observable below 1,000 cycles, and it is concluded, therefore, that the pulsations of the vas spirale are ineffective in producing appreciable changes in sensation over the distal half of the basilar membrane. It is to be remarked that at higher intensities the heart beat normally produces no perceptible fluctuation in intensity, whereas it would seem that it should be greater. The phenomenon here noted probably has a more deep-seated cause associated with the intimate mechanism of stimulation of the nerve. The remarks about it must therefore be taken as suggestive speculation.

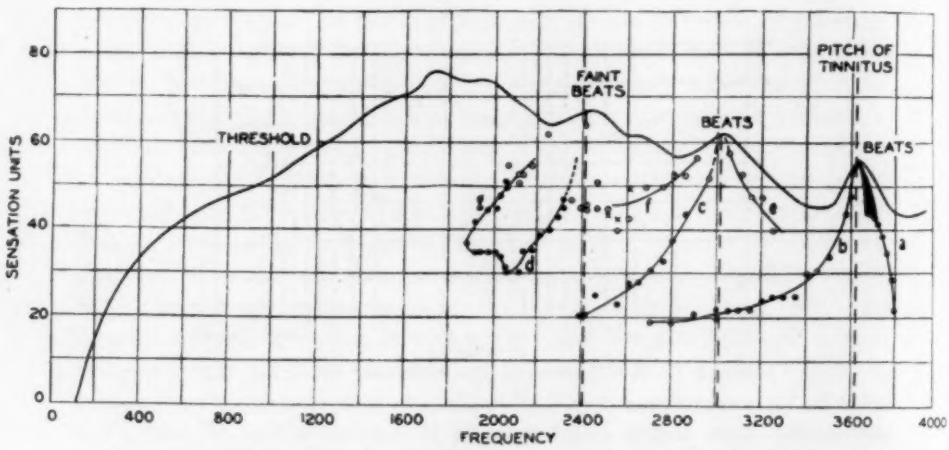

Chart 2.—Analysis of tinnitus of an apparent pitch of 3,620 cycles; May, 1928; left ear of R. L. W.

The test tones used sounded like perfectly pure, smooth, sustained tones, like those from a tuning fork, except when the intensity and pitch were such as to bring them within the irregularly shaped area on the diagram (chart 2), that is, between frequencies 1,900 and 3,800 cycles, and for intensities within 40 sensation units of the threshold. Within this abnormal range, the pure tone incident on this ear was perceived as being impure and discordant. The exact nature of this discord varied from one place to another. This distortion of the perception of a pure tone is due to the presence of tinnitus, the nominal or apparent pitch of which is 3,620 cycles.

The significance of this diagram will be better understood from a detailed description of the data that it represents. Without a tone in the telephone receiver the only sound heard was, of course, the tinnitus.

If a tone of frequency, say 3,400, was introduced at a very faint intensity and then gradually increased, its appearance at the threshold, corresponding to a reading of the attenuator of 46, was marked by an apparent change in the character of the tinnitus. As the intensity of this tone is further increased its combination with the tinnitus produced a definite discord which was most pronounced at an intensity 35, but disappeared abruptly at 30. For sounds of louder intensity than this the tinnitus was entirely masked and the tone sounded perfectly pure. The point 30, it will be seen, is on curve *b*, which represents the sharply defined boundary, in the frequency range of from 2,700 to 3,600 cycles, between a discord above and a pure tone below and is, in other words, a part of the "masking" curve of the tinnitus. Curve *a* represents the same boundary for tones higher than 3,600 cycles. The dots represent measured values.

When the tone was of a pitch of 3,620 cycles, its threshold was marked by the appearance of an unmistakable "beat" with the tinnitus. These beats were not as regular and smooth as the mechanical beats produced by two tuning forks, but consisted definitely of a fluctuation in intensity of the perceived sound, which might be described as "mushy" beats. As the test tone was slowly decreased in pitch, the beats perceptible at or near the threshold became faster and resulted finally in the discord described for 3,400 cycles, the disappearance of which is marked by curve *b*. When, however, the pitch was somewhat higher than 3,620, say 3,660, the appearance of the tone was again marked by fast beats at the threshold, but when the intensity was increased into the range of from 45 to 52 a complete silence resulted. Any sound of pitch and intensity represented by the black area between 3,600 and 3,200 cycles not only rendered the tinnitus inaudible, but was itself extinguished. Experimentally, the boundaries of this dark area were marked by an abrupt transition from sound to silence. Except for this silent area, curves *a* and *b* might easily, without the tinnitus present, represent the masking curve of a sound of pitch 3,620 and of intensity about 10 sensation units above the threshold.

When a tone of frequency 2,800 cycles was used, its appearance at the threshold, about 57, was apparent by the appearance of a discord. As this intensity was increased, a definite change in its nature occurred at 53. Further increase produced a gradual change in the nature of this discord until the point 38 on curve *e* was reached, where a second and more marked change occurred in the character of the discord. When the intensity was further increased to 19, the discord disappeared abruptly, giving place to the perception of a pure tone, a condition again for complete masking of the tinnitus.

With this description the data represented by curves *a*, *b*, *c*, *d*, *e*, *f* and *g* will be clear. In all cases the dots represent transitions which are

abrupt and accurately located. The circles represent transitions which are less abrupt, but quite definite. The crosses represent more gradual transitions which are much less definite and are likely on repetition of the measurement to be missed or to be located elsewhere. The actual measurements contain many points representable by crosses, but were not plotted. It was found that the threshold varied considerably more from day to day than did the boundaries represented by the other curves. In addition to this the threshold was at all times much less sharply marked than the transitions.

One point further needs to be noted to describe the data. At a pitch just over 3,000 cycles beats were again plainly audible near the threshold, but not quite as distinct as those at 3,620. The same beat phenomenon occurred at 2,400 cycles, but not so near the threshold and much less distinct than at the two higher frequencies. Although a careful search was made throughout the frequency range of from 100 to 15,000 cycles, no other beat points or distortion was discovered.

The corresponding diagram for the other ear was quite different, and the boundary lines were less distinct. I have recently repeated the measurements on the left ear and found curves *a* and *b* still well marked, but only a part of *c* remained. The rest seem to be more or less obliterated. This ear has changed markedly during the past two and a half years. The tinnitus, however, remains and at approximately the same pitch. It must therefore have changed in character or intensity. My impression is that it has become more oppressive, but such an impression is of course entirely untrustworthy as data.

There are a number of experimental facts shown by this diagram (chart 2) to be noted:

1. The distortion occurs only at low intensities near the threshold. This is opposite to the so-called "nonlinear" distortion which occurs at higher intensities and increases rather than diminishes with intensity. This circumstance is fairly convincing, though not absolute, evidence that the tinnitus is not of mechanical origin.

2. The distortion occurs within a small frequency range of one octave, from 1,900 to 3,800 cycles, which, with reference to chart 1, it will be seen is at the brink of a sharp drop below the normal of the acuity curve. This suggests that at the time of the first tests the nerve tissue associated with the organ of Corti was partially atrophied along the nominal length of from 2 to 6 mm. and that the disease was progressing along the membrane, being then in active process of degeneration in the range of from 6 to 10 mm. from the proximal end of this organ.

3. There are three frequencies in the distortion range which distinguish them in contrast with any others. In the order of their

prominence they are 3,600, 3,000 and 2,400, each being approximate. They are marked (*a*) by prominences in the threshold curve, (*b*) as convergent points for the sensation transition boundary curves (*a, b, c, d, e, f* and *g* of chart 2) and (*c*) by beats.

Notice that these frequencies are the fourth, fifth and sixth harmonics of 600 cycles and that the first three harmonics, 600, 1,200, 1,800, are missing, or at any rate imperceptible. This is a second strong indication that the origin of the tinnitus is not mechanical, for while it is theoretically possible to have a vibration of such a wave form, its random occurrence in nature or even in the arts is improbable. It is more reasonable to suppose that the beats at these three points are due to interference, in generating nerve impulses, between the periodic sound stimulus and the autoperiodic chemical or electric stimulus of the agent of tinnitus. If this is the correct explanation, it follows that the normal frequency of discharge of a single nerve fiber in this ear in response to this "chemical" stimulus is 600 cycles. This is not in bad agreement with the estimate by Professor Forbes, of Harvard Medical School, of 800 cycles for the maximum possible rate of discharge of such a sensory nerve fiber. It is supposed by neurologists that this rate increases with the intensity of the stimulus up to a certain maximum value which cannot be exceeded. This frequency of maximum impulse varies from one nerve to another and even among single nerve fibers of any nerve trunk, being highest in the sensory nerves. This in itself is a noteworthy circumstance, for it must be related to the exigencies of the different functions.

Viewed in this light, it is evidently a remarkable coincidence that in this ear the seat on the organ of Corti of the tinnitus should happen to be almost exactly at a spot where a sound stimulus having a frequency of some even multiple of 600 also stimulates it. It is too long a story to go into the possibilities of variation in form of the tinnitus masking curves which might be expected. My other ear gives a different diagram, and the few random observations made on ears of other subjects indicate that chart 2 is a rather unique condition of the ear.

If the foregoing explanation is correct, it will be seen that although the apparent pitch of the tinnitus is 3,600 cycles per second, the actual frequency of discharge of the separate nerve fibers is a matter of 600. The indications are that the apparent pitch of the sound is dependent primarily on the place on the membrane where it is received rather than on the frequency of the impulse traveling over a single nerve fiber. This does not, of course, preclude the possibility of a 3,600 cycle component in a large number of staggered 600 cycle impulses sent over a bundle of nerve fibers situated close together. In fact, the recent

experiments of Wever and Bray [3] at Princeton show clearly that such is the case.

It is a consequence of the qualitative theory of frequency selection by the basilar membrane that the stimulus on separate fibers should be staggered. According to this, the whole membrane vibrates at any one frequency, but with greater amplitude at a certain place than on either side. On the proximal side of this spot, such minute vibration as exists is in phase with the motion of the stapes, because the only appreciable reaction is that of the elasticity of the membrane, the inertia of the fluid having little effect. On the far distal side of the active spot, however, the inertia of the fluid controls and such small motion as exists is in opposite phase to the motion of the stapes. The phase of the vibration relative to that of the stapes should change gradually in passing through the active area. In other words, the small motion of the proximal end is in step with the stapes; at the center of the active area, it is a quarter step behind and at the distal end, a half step behind. If it is true that each vibration of a hair cell constitutes a separate stimulus, or at least an appreciable component of a separate stimulus, it is obvious that each vibration must produce a succession of stimuli progressing along the membrane but with a maximum of intensity in the so-called active region where the vibration is greatest. The only nerve fibers responding will be some of those near the active area, where the amplitude of the vibration is above the threshold value. If the frequency of the vibration is much lower than the highest (from 600 to 800 cycles) that it is possible for a single fiber to transmit, it would be expected that each vibration would send a volley of staggered impulses up the auditory nerve which, when regarded as a whole, would constitute one large composite impulse on this trunk. This composite impulse occurs once for each vibration, and it is this impulse that was picked up and observed by Wever and Bray. Their experiments indicate that the frequency of the impulse is the same as that of the stimulus, so it would appear that a vibration is effective as a stimulus only once as a complete cycle. It would further be expected that an oscillographic record of this composite impulse would not be the same as that on a single fiber magnified. It would also be expected that this wave of action potential thus picked up from the nerve trunk in response to a pure tone should be an impure tone. The impurity should increase as either the frequency or the intensity of the stimulus is lowered, not markedly, however, because of the smoothness of the separate impulses.

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3. Wever and Bray: The Nature of Acoustic Response: The Relation Between Sound Frequency and Frequency of Impulses in the Auditory Nerve, J. Exper. Psychol. **13**:373 (Oct.) 1930.

The purity of a wave of action potential in response to a pure tone stimulus is measured by the degree of its resemblance to a sine wave. If only one nerve fiber responded, the purity would decrease as the frequency was lowered, for then each cycle of the action potential would be represented by only one impulse. At moderately high intensities, a large number of fibers respond, and owing to the staggering action of the stimulus, the composite impulse should be a better approximation to a smooth wave. At very high intensities, where all fibers over an appreciable range of the organ of Corti are brought into play, there should be no possibility of further increasing the peak value of the action potential wave, and any addition must be due to the addition of new impulses on its sides. Thus, in this case, the wave of action potential should be "flat top." It is to be expected, therefore that there is a distinct limit to the possible range of intensities of the action potential wave and that its purity should be greatest at moderate sound intensities, but not an exact copy of the sound wave in any case. This is in accord with the conclusions arrived at by Fletcher,[4] before the results of Wever and Bray were available, by a somewhat different process of reasoning.

In the study of action potentials of nerve trunks, a stimulus in the form of an electric shock, or a series of shocks, is applied at some place on the nerve trunk. This method of stimulation presumably stimulates all of the component fibers simultaneously. Thus, when the frequency of application of the stimulus is increased, the highest frequency at which the action potential follows the stimulus is the same for the trunk as for a single fiber. If the foregoing reasoning is correct, it would be expected that if a nerve trunk, say, for example, the frog's sciatic nerve, could be split and the two branches thus obtained be separately stimulated in staggered phase, the highest frequency of the action potential of the trunk should be double that obtainable when the stimulus is applied, as is usual, to the whole trunk. Similarly, if the nerve trunk below the point where the stimulus is applied could be split into three branches and each branch could be stimulated by separate shocks staggered in phase, it should be possible to obtain action potentials on the trunk of a frequency three times that ordinarily given for the same nerve. I understand that this experiment has never been tried, but it should be in the nature of a crucial test for the foregoing reasoning.

Bell Telephone Laboratories, Inc.

4. Fletcher: Spare Time Theory of Hearing, J. Acoustical Soc., America **1**:311 (April 30) 1930.

THE LARYNX IN PARALYSIS OF THE RECURRENT LARYNGEAL NERVE*

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Two questions have long retained their interest for the laryngologist: 1. Why does adduction instead of abduction of the vocal cords follow section of the inferior laryngeal nerves? 2. After the paralyzed vocal cords are in adduction, what can be done to help the patient? In an attempt to see whether any further light could be thrown on these problems, the research here reported was undertaken.

MUSCLES AND NERVES OF THE LARYNX

In man, the intrinsic muscles of the larynx may be divided into two groups: those that abduct and those that adduct the vocal cords.

The abduction of the vocal cords is dependent on the posterior crico-arytenoid muscle. This muscle separates the cords, and consequently opens the glottis by rotating the arytenoid cartilages outward around a vertical axis, passing through the crico-arytenoid joints, so that the vocal processes and the vocal cords attached to them become separated.

The muscles that control adduction are the cricothyroid, the crico-arytenoid lateralis and the thyro-arytenoid. The interarytenoid, an unpaired muscle situated between the two arytenoid cartilages, also helps in producing adduction. The lateral crico-arytenoid muscles close the glottis by rotating the arytenoid cartilage inward so as to approximate the vocal cords. The interarytenoid muscle approximates the arytenoid cartilages, and thus closes the opening of the glottis, especially of the posterior part.

The cricothyroid muscle produces tension on the vocal cords, causing elongation. This is brought about by the following movement: The thyroid cartilage is fixed by its extrinsic muscles, then the cricothyroid muscle draws the cricoid cartilage upward, depressing the posterior portion of the cricoid cartilage, which is attached to the arytenoid, thus stretching the cords.

The thyro-arytenoid muscle consists of two parts, each having a different attachment and direction. The action of these two parts, therefore, is rather complicated. Their main function is to approximate

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the arytenoid and thyroid cartilages and thereby relax the vocal cords. Owing to the connection of the inner portion of the muscle with the vocal cord, this part is supposed to modify the elasticity and tension of the cord, and the outer portion, being inserted into the anterior surface of the arytenoid cartilages, may rotate the cord inward and thus narrow the rima glottidis by bringing the cords together.

The innervation is derived from the superior laryngeal and inferior laryngeal nerves, together with branches from the cervical sympathetic nerve. The superior laryngeal nerve divides into the external and the internal laryngeal nerves, the former of which is the motor nerve to the cricothyroid muscle, while the latter, which is mainly sensory, supplies the interior of the larynx. Some motor fibers are said to be carried to the interarytenoid muscle. The sensory branch communicates with a branch from the inferior (recurrent) laryngeal nerve, the nerve that supplies all the other muscles of the larynx. Sympathetic fibers accompany all of the laryngeal nerves, but their exact function is unknown at present.

Paralysis of either recurrent laryngeal nerve deprives all the intrinsic muscles of the larynx of their function on the side that is stricken, with the exception of the tensor muscle of the vocal cord, which is supplied by the external branch of the superior laryngeal nerve. This leads to inactivity of the vocal cord on the paralyzed side. The cadaveric or midposition is thought to result from the action of the cricothyroid muscle, which is supplied by the external branch of the superior laryngeal nerve. In slow paralysis of the recurrent nerve, the abductor muscle of the larynx is affected first. Many theories have been offered for this phenomenon, but as yet few have much evidence to support them.

THEORIES ADVANCED TO EXPLAIN PARALYSIS OF ABDUCTION

Rosenbach [1] was one of the first to recognize the uniformity with which the abductor muscle of the larynx is affected, and he announced that the fibers of the recurrent nerve which dilate the rima glottidis possess a greater vulnerability than those which narrow the glottis. Semon [2] pursued the research further, and expanded Rosenbach's theory to include a like vulnerability along the whole central and peripheral course of the recurrent nerve. He formulated the law that in all organic injuries affecting the recurrent nerve in its central or peripheral fibers, the abductor fibers are the first attacked. This statement has long

1. Rosenbach, O.: Ist der Satz von der verschiedenen Vulnerabilität der Recurrensfasern berechtigt? Arch. f. Laryng. u. Rhin. **6**:588, 1897.

2. Semon, F.: On the Position of the Vocal Cords in Quiet Respiration in Man and on the Reflex Tonus of Their Abductor Muscles, Proc. Roy. Soc., London **48**:403, 1890.

been recognized as incorrect so far as it refers to paralysis of central origin; however, when applied to peripheral lesions, it is essentially correct.

Hooper [3] stated that the special vulnerability of the abductor fibers could be explained by the importance of the two laryngeal functions, phonation and respiration. He thought that closure of the larynx to prevent the aspiration of foreign bodies explains the persistence of the adduction in paralysis of the vocal cords, since this is due to the maintenance of protective mechanisms.

From experiments on dogs, Russell [4] concluded that the abductor and adductor fibers in the recurrent nerve were divided into several bundles, the one group separate and distinct from the other, and each maintaining its course throughout the length of the nerve from its origin to its termination. He also reported success in causing abduction or adduction of the vocal cord, according to the nerve bundles that were stimulated. These findings have never been substantiated. In the laboratory of the Cleveland Clinic Foundation, the nerve has been studied carefully according to the method devised by Russell, but my associates and I have not been able to duplicate Russell's results.

Krause [5] considered that the adduction resulting from lesions of the recurrent nerve was due to a muscle cramp of all the muscles supplied by the nerve, and as there is a preponderance of adductor muscles, adduction results. Simanovski [6] has endeavored to discover the reasons for the varied vulnerability of the laryngeal muscles by a study of their difference in anatomic structure. He assumed that the posterior crico-arytenoideus was a smooth muscle and drew an analogy from Grabower's work on comprehensive histologic studies. Grabower [7] found that the posterior crico-arytenoid muscle is poorly innervated in comparison with the other laryngeal muscles supplied by the inferior (recurrent) laryngeal nerve. According to Broeckaert,[8] the sympathetic innervation plays a part in the production of paralysis of the recurrent nerve.

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3. Hooper, F. H.: The Anatomy and Physiology of the Recurrent Laryngeal Nerves, New York M. J. **46**:29, 63, 99, 150 and 188, 1887.

4. Russell, J. S. R.: The Abductor and Adductor Fibers of the Recurrent Laryngeal Nerve, Proc. Roy. Soc., London **51**:102, 1892.

5. Krause, H.: Ueber die centrale motorische Innervation des Kehlkopfes, Berl. klin. Wchnschr. **27**:81 and 110, 1890.

6. Simanovski, N.: O kolebaniyakh golosovikh svyazok pri paralichakh razlichnikh gortannikh mishts, Ejened. klin. gaz. **7**:509, 1887.

7. Grabower, H.: Bemerkungen zur zentralen Kehlkopfmuskellähmung und zum Rosenbach-Semonschen Gesetze, Arch. f. Laryng. u. Rhin. **23**:57, 1910.

8. Broeckaert, J.: Zur Kenntnis der Veränderungen in den Kehlkopfmuskeln nach Durchschneidung des Rekurrens, Arch. f. Laryng. u. Rhin. **21**:453, 1908-1909.

Bilancioni and Tarantelli [9] have shown that ablation of the adventitia of the carotid artery immediately provokes an intense anemia of the vocal cord and the internal surface of the glottis. They thought that following this procedure the vocal cords showed a slight paralysis and a general loss of tonus. Leriche and Fontaine [10] reported that in the Strasbourg Clinic the larynx was studied in several cases in which cervical sympathectomy was performed because of disorders involving the arm. They stated that following ramisectomy a marked hyperemia of the pharynx and the nasal mucosa occurred, but that the vocal cords did not seem to be involved. In the Cleveland Clinic, we observed the larynx in the following case, in which cervical sympathectomy was performed.

REPORT OF CASE

A woman, aged 34, came to the clinic complaining of pain in the left arm which extended from the axilla to the tips of the fingers, with a sensation of numbness and tingling along the distribution of the ulnar nerve. Shortly after the onset of the pain the patient had noticed a swelling in the left side of the neck, which was thought to be in the left lobe of the thyroid gland. The patient felt that the muscle power of the left arm was diminishing, and noted a tendency of this arm to tremble more than the right. Occasionally, she had a mild sensation of choking. An estimation of the basal metabolism as well as a clinical examination did not show evidence of hyperthyroidism.

On physical examination, the one important finding was a tumor in the left side of the neck in the region of the left lobe of the thyroid gland. This mass was firmly fixed and slightly tender on pressure. The trachea appeared to be displaced toward the right. The tingling and numbness in the left arm were still present. There was also pain along the distribution of the ulnar nerve and weakness of the flexor muscles of the left arm.

Examination of the vocal cords is made routinely in all cases in which surgical intervention is performed on the neck, and laryngeal examination in this case revealed that while there was both adduction and abduction of both cords occasionally the left cord was temporarily held in spasm but soon relaxed. The cords were reexamined on the following day and appeared to be functioning normally.

At operation, a hard, white, ovoid tumor about the size of a hen's egg was found posterior to the carotid sheath which with its contents was displaced to the right. The left lobe of the thyroid gland and the trachea also were displaced to the right. The tumor was firmly fixed posteriorly to the body of the sixth cervical vertebra. Anteriorly and laterally, it was covered by a capsule of thin connective tissue. At two points on its anterior surface the cervical sympathetic ganglions were identified. The tumor was excised, and the cervical sympathetic chain was removed. During this procedure, the left half of the face and neck was flushed, while the right half was pale. The left eye showed enophthalmos,

9. Bilancioni, G., and Tarantelli, E.: Laringe e simpatico; Nota preliminare sull' innervazione simpatica della laringe, Arch. ital. di otol. **33**:321, 1922.

10. Leriche, R., and Fontaine, R.: De quelques phénomènes observés après la section des derniers rameaux communicants cervicaux et du premier dorsal, J. de chir. **27**:143, 1926.

narrowing of the palpebral fissure and marked reduction of the pupillary diameter. This group of symptoms is commonly seen when section of the cervical sympathetic ganglions is made.

The day after operation a laryngeal examination was made. Apparently, the function of the vocal cords was not disturbed, and they moved normally in abduction and adduction.

EXPERIMENTAL WORK REPORTED

In his experiments on dogs, d'Onofrio [11] noted that stimulation of the cervical sympathetic trunk with a galvanic current caused a spasmodic contraction of the vocal cords. Under the influence of a faradic current, the cord was in strong abduction, and he attributed this to direct sympathetic stimulation. In all of his experiments, ischemia of the mucosa was noted. D'Onofrio's histologic studies of the laryngeal muscles from thirty-five to fifty days after ablation of the ganglions showed marked atrophy of the glands of the larynx, but little modification of the other tissues.

On the basis of his studies of the effect of the sympathetic nerves on the larynx, Kakeshita [12] stated that he observed no effect on the laryngeal muscles. He felt that the sympathetic fibers ended in the vessels of the larynx.

Marx [13] studied the relation of the sympathetics and the larynx by means of drugs and section of the cervical sympathetic chain. He found that atropine, which paralyzes the craniosacral sympathetics, had no influence on the function of the recurrent nerve. He likewise found that ergotamine had no effect on the laryngeal muscles. Yamamoto [14] found that section of the sympathetic nerves causes dilatation of the vessels supplying the muscles of the larynx.

EXPERIMENTAL PRODUCTION OF AN ACUTE CONDITION

An acute condition was produced in a dog under morphine-ether anesthesia; a midline incision was made in the neck, exposing the trachea and larynx. The nerves of the larynx were easily identified, and were stimulated by a faradic current. The vocal cords were observed by direct vision through the animal's mouth, and the influence on them of stimulation of the various nerves was determined.

Stimulation of the right recurrent laryngeal nerve caused adduction of both vocal cords in the midline, and closure of the interarytenoid space was complete.

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11. d'Onofrio, F.: Esperimenti e considerazioni sull'influenza del simpatico pericarotideo sulla laringe, Arch. ital. di otol. **35**:129, 1924.

12. Kakeshita, T.: Kehlkopf und Sympathicus, Arch. f. d. ges. Physiol. **215**: 22, 1926.

13. Marx, H.: Untersuchungen zur Innervation des Kehlkopfes, Acta otolaryng. **12**:45, 1928.

14. Yamamoto, J.: Untersuchungen über den Einfluss der sympathischen Innervation auf die Permeabilität der Gefässe, Biochem. Ztschr. **145**:201, 1924.

The vocal cords remained closed as long as the stimulation was continued. Under deep anesthesia, the same result was obtained, although it was necessary to use a stronger current.

Stimulation of the vagus nerve on the right side caused the cords to approximate in the midline, and at the same time respiration ceased.

Stimulation of the cervical sympathetic fibers just as they left the brachial plexus caused a fluttering action of the vocal cords, but no decided abduction or adduction. It is interesting to note that during the stimulation of the cervical sympathetic chain, the eye on the stimulated side protruded, presenting an appearance not unlike that of a unilateral exophthalmos. The palpebral fissure was widened, and the pupil of the eye was greatly dilated. When stimulation of the eye was not observed, there was no action of the vocal cords.

Stimulation of the intact superior laryngeal nerve interrupted respiration, and this was followed by a swallowing movement. Stimulation of the external branch increased the tension of the vocal cord on the same side, because of the elongation of the cord in an anteroposterior direction. Neither division of the superior laryngeal nerve nor stimulation of the peripheral end of the nerve produced any change in the action of the vocal cords. Stimulation of the central end of the nerve caused adduction of the cords and cessation of respiration. At the end of stimulation, a swallowing movement occurred.

Stimulation of the peripheral end of the divided recurrent laryngeal nerve was followed by adduction of the vocal cord on the same side, the cord on the opposite side continuing to function normally. The central end of the nerve did not give a response to stimulation.

In a second animal, under morphine-ether anesthesia, the neck was opened and the various nerves were stimulated in order to identify them and record their normal action. In this dog the cervical sympathetic chain on the right side was divided. There was no observable change in the action of the vocal cords. Stimulation of the peripheral end of the cervical sympathetic chain resulted in a spasmodic contraction of the vocal cords, while stimulation of the central end caused great pain, but a change was not observed in the vocal cords. On sectioning the cervical sympathetic chain near its origin, enophthalmos, ptosis and miosis of the eye on the same side were observed.

Experiments in which an acute condition was produced were repeated many times, small variations being made in the procedures, but the results were always uniform. At no time was abduction of the vocal cords produced by artificial stimulation of any nerve. Stimulation of the recurrent nerves caused adduction, and when they were cut the cords ceased to act and assumed a position in the midline of the larynx. Activation of the cervical sympathetic nerve caused a fluttering of the vocal cord on the side stimulated, which tended to keep the cord in adduction. Because of the proximity of the cervical sympathetic nerve to the vagus nerve in the dog, it was thought that possibly a spread of the current to the vagus nerve may have caused the adduction of the cords.

EXPERIMENTAL PRODUCTION OF A CHRONIC CONDITION

In order to study the action of the various nerves on the vocal cords more accurately, chronic conditions were produced in dogs and the animals were allowed to recover.

On Dec. 1, 1928, a large black police dog was operated on, the right cervical sympathetic chain being cut low in the neck near its connection with the vagus nerve. After the operation the right eye showed a typical Horner syndrome. The vocal cords were not changed in function or appearance. The dog was examined periodically for the next three weeks, and no change in the action of the vocal cords was ever observed.

On December 26, the dog was again operated on, and the right vagus nerve was stimulated with an interrupted faradic current. The vocal cords were adducted, but there was a small space about 1 mm. wide between them. Stimulation of the left vagus nerve caused complete adduction, with no space remaining between the two cords. Stimulation of the right recurrent laryngeal nerve resulted in adduction of both vocal cords in the midline, while stimulation of the left induced strong adduction. It appears, therefore, that section of the sympathetic chain on the right side causes a slight paresis of the vocal cord on that side. When the superior laryngeal nerve on either side was stimulated, the results were identical with those in the intact animal.

On Feb. 20, 1929, a third operation was performed on this dog, the right recurrent laryngeal nerve being sectioned. The right cord was then functionless and came to rest a few millimeters to the right of the midline. The left cord continued to function.

The dog was observed again in June, 1929. At this time there was paralysis of the right vocal cord, the cord lying in the mid or so-called cadaveric position. The left cord continued to function, and when stimulated became adducted across the midline so that it met the paralyzed right cord and succeeded in closing the larynx. The appearance of this dog's larynx was the same as that following a simple section of the recurrent laryngeal nerve.

In July, the dog was killed. On gross examination of the larynx, atrophy of the muscles on the right side was apparent. Few or no muscle fibers were present in the abductor of the right vocal cord. The adductors were also atrophied, but to a lesser degree. The muscles of the left cord did not show gross pathologic changes, but had a normal, healthy appearance. The larynx was then dissected free, and its muscles were saved for histologic study.

On Dec. 26, 1928, both cervical sympathetic nerves of a large collie (dog 2) were cut. This dog had a bilateral enophthalmos, ptosis of the upper eyelid and miosis of the pupils. Examination of the larynx following the operation showed the vocal cords to be functioning normally. On Jan. 9, 1929, the cords did not show a change in appearance or function. The dog was killed, and the larynx was saved for histologic study.

On Sept. 25, 1928, dog 3, a small airedale, was operated on, both recurrent laryngeal nerves being sectioned. The cords which were separated for a distance of approximately 0.25 cm. were paralyzed and exhibited no motility. Examinations were made from time to time, and the cords were found to vary little in their position. The dog was able to breathe without difficulty when at rest, but when he exercised or became excited, dyspnea developed. This dyspnea was of an unusual type, the difficulty being expiratory rather than inspiratory. On Jan. 21, 1929, the right cervical sympathetic chain was cut, but this operation was not followed by any appreciable change in the position of the vocal cords. On February 14, the cords were again examined and no further change was noted, except that the right vocal cord appeared to show a greater degree of atrophy than the left.

Dog 4, a large coach dog, was operated on on Sept. 4, 1928. The right superior laryngeal nerve was cut, and the dog was allowed to recover. Observation of the

vocal cords revealed no change in function or appearance. At times the dog had a harsh cough, but dyspnea was not apparent.

On October 22, the right recurrent laryngeal nerve was cut. Following this operation the right vocal cord was just lateral to the midline and was motionless. The dog was again examined on Feb. 2, 1929. At this time the right cord was immobile, had deviated from the midline a few millimeters and was atrophied considerably. The left cord was functioning normally, and at times approximated the edge of the right cord. In June, the dog was killed, and a histologic study was made of the muscles of the larynx.

Dog 5, a small pup, 2½ months old, was operated on on Jan. 29, 1929. Both recurrent laryngeal nerves were sectioned, after which the dog had some difficulty in respiration, being unable to fill the lungs with air. Neither vocal cord was motile. The cords were approximated in the midline, a narrow aperture a few millimeters in width permitting respiration. On February 5, the dog caught cold and had considerable difficulty in breathing. In the afternoon he stopped breathing and tracheotomy was performed, but he died during the night. On examination of the larynx, the vocal cords were found to be firmly adducted in the midline. The larynx was composed mainly of soft cartilage, and its probable collapse was due to the strong action of the muscles.

In dog 6, an old bulldog, both recurrent laryngeal nerves were sectioned on Feb. 2, 1929. The vocal cords were immobile on examination and were adducted almost to the midline. This dog had no difficulty in breathing, however, unless he became excited or exercised vigorously, when an inspiratory dyspnea developed.

HISTOLOGIC STUDIES

When only the recurrent laryngeal nerve was sectioned, atrophy of all the muscles on the corresponding side of the larynx was observed. In one animal the left recurrent laryngeal nerve was sectioned. Two months later, microscopic sections were made of the muscles of the larynx. The atrophy of the abductor fibers of the left vocal cord was much greater than that of the adductor fibers of the same cord. Atrophy of the muscles on the opposite side of the larynx was not present in any case.

In the larynx of another dog, in which the right superior laryngeal nerve and the right recurrent laryngeal nerve were sectioned, the muscles on the right side only were affected. The degree of atrophy was much greater in this dog's larynx than in the larynx just described. It appears from this study that while the superior laryngeal nerve does not affect the motor function of the laryngeal muscles, it does exert a trophic effect on them.

In one case in which the sympathetic ramus was sectioned proximal to its connection with the vagus, the muscles were studied microscopically. There was no histologic change in either the abductor or the adductor group of muscles of the larynx. In another animal the sympathetic ramus and the recurrent laryngeal nerve were sectioned on the same side. In this dog the larynx appeared the same as one in which a simple section of the recurrent laryngeal nerve had been done. His-

tologically, the muscles showed about the same degree of atrophy that might be expected if only the recurrent laryngeal nerve had been sectioned.

From the histologic studies it appeared that a greater degree of atrophy occurred in the abductor than in the adductor fibers following section of the recurrent laryngeal nerve.

ANASTOMOSIS OF VARIOUS NERVES OF THE NECK WITH THE RECURRENT LARYNGEAL NERVE

Colledge and Ballance [15] reported a series of experiments in which an attempt was made to anastomose the cut end of the recurrent laryngeal nerve with various other motor nerves in the neck. Since it has been shown that an anastomosis between the spinal accessory and the seventh nerve may result in a return of the function of the facial muscles, the authors decided to use other motor nerves in the neck. Accordingly, they experimented with anastomosis of the phrenic nerve with the recurrent, the recurrent with the vagus and the recurrent with the descendens noni, a branch of the ansa hypoglossi. The anastomosis was done by either of two methods, the end-to-end or the end-to-side. The authors reported that in a few cases they were able to obtain a return of function when the animals were placed under partial asphyxia and hyperpnea developed. In none of the animals was normal function obtained.

Negus [16] stated that the impulses that traverse the various nerves of the neck are synchronous with the opening and closing of the larynx. He showed that the impulses traveling to the diaphragm are not synchronous with those going to the abductor muscles of the larynx. He predicted that even though the anastomosis were functioning, it would result in an incoordinate action of the respiratory organs as a whole.

Blalock and Crowe [17] reported a series of experiments in which an anastomosis was made between the vagus and recurrent and the recurrent and descendens noni, and reported a return of function of the laryngeal muscles in a majority of cases.

EXPERIMENTAL DATA

In a series of five dogs, various methods of anastomosis were tried. The recurrent nerve was anastomosed with the phrenic nerve in two

15. Colledge, L., and Ballance, C. A.: The Surgical Treatment of Paralysis of the Vocal Cord and of Paralysis of the Diaphragm, Brit. M. J. **1**:553 and 609, 1927.

16. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.

17. Blalock, A., and Crowe, S. J.: The Recurrent Laryngeal Nerves in Dogs: Experimental Studies, Arch. Surg. **12**:95 (Jan.) 1926.

cases. In one instance there was an anastomosis between the phrenic nerve and the external branch of the superior laryngeal nerve, and also between the ansa hypoglossi and the peripheral end of the phrenic nerve. In two animals an anastomosis was made between the peripheral end of the recurrent nerve and the descendens noni. These operations were performed on March 22, 1929.

Examinations were made at various intervals, and at no time was any return of function observed. The dogs were killed in September, 1929, 160 days after the operation. The neck of each was opened and the point of anastomosis found. Stimulation of the nerve trunk on the central side of the anastomosis failed to produce any motion of the vocal cords, which had assumed the midposition. Histologic studies showed atrophy of the laryngeal muscles on the side of operation.

COMMENT

It appears that if anastomosis is to be of use clinically the results of experiments should be more encouraging. The successful anastomoses reported here have been confined, as a rule, to a small percentage of the total number of operations performed. Colledge and Ballance described one case in which the return of function was questionable. Another obstacle is the difficulty of finding the divided ends of the laryngeal nerve after it is sectioned. Negus determined, by means of the action current, that the opening of the glottis and the descent of the diaphragm are not synchronous, and that therefore dyspnea and incoordination of the muscles of respiration would result if the anastomosis did function.

EFFECT OF DRUGS ON THE LARYNGEAL MUSCLES

Since the recurrent laryngeal and the superior laryngeal nerves are both branches of the vagus, it was thought that various drugs might affect the mechanism of the larynx. In addition, since sympathetic fibers have been found to enter the larynx, drugs which affect the sympathetic nerves were also used.

Under morphine-ether anesthesia, a midline incision was made in the neck, and the trachea and larynx were exposed. A tracheotomy tube was placed in the trachea low in the neck. A soft rubber balloon was then placed between the vocal cords, and the balloon was connected with a tambour. By this method the abduction and adduction of the vocal cords could be studied.

When 1 cc. of epinephrine (1 : 1,000) was injected subcutaneously, the excursions of the cords appeared to be of greater amplitude and the adduction was much greater on expiration, while the abduction apparently was unaffected. Several minutes after the subcutaneous injection of 1 mg. of atropine, the excursions of the vocal cords appeared to be

slightly decreased in amplitude but increased in number. The injection of 5 mg. of pilocarpine induced a profuse secretion of saliva, but no change in the motility of the vocal cords.

Many organs, including the larynx, that are innervated by the vagus and the sympathetic nerves have been shown to be controlled to some degree by a mutual antagonism between the two. The larynx, however, differs from most of these organs, since it has the function of phonation, which is a voluntary mechanism. From a study of the effect of drugs on the muscles of the larynx, no antagonism of the two divisions of the autonomic nervous system could be demonstrated.

SUMMARY

Experiments are described which were carried out in an attempt to determine the cause of adduction of the vocal cords following section of the inferior laryngeal nerves, and the effect on bilateral paralysis of the recurrent nerves of anastomosis of various nerves to the recurrent laryngeal nerve and also of certain drugs. It is apparent that while the larynx does have a dual innervation through the vagus and the thoracic sympathetics, the two are not antagonistic in their action, and that cervical sympathectomy causes no change in the motor activity of the cords.

The application of drugs known to act on one division of the autonomic system does not cause any predictable change in the action of the vocal cords. Atropine does not produce a paralysis of the craniosacral sympathetic fibers, and epinephrine likewise fails to stimulate the vocal cords by acting on the cervical sympathetic fibers.

The experiments in which anastomosis of the nerves was uniformly unsuccessful demonstrate that the feasibility of this operation in human beings is slight.

THE SYMPTOM OF VERTIGO*

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Many and varied are the feelings and sensations described as vertigo, but in general they fall into one or the other of two main patterns: the rotatory or turning vertigos and the tactile vertigos. As turning vertigo is nearly always a sign of disturbed vestibular function and tactile vertigo may also at times be of vestibular origin, it is fitting that they should peculiarly interest members of our specialty.

The vestibular mechanism has two distinctive functions or reactions. These reactions are vertigo and nystagmus, and whenever a vestibular mechanism is irritated these reactions can be obtained. The vestibular mechanism may also concern itself with other reactions, such as past pointing and disturbances in equilibrium, but it does not actually cause these reactions in the same way in which it causes nystagmus and vertigo, for they are the distinctive reactions of this mechanism.

Turning or rotatory vertigo is the most certain sign of vestibular invasion, peripheral or central. In this connection, I wish to take exception to a statement made by Dr. George McKenzie [1] in an article that appeared in the 1930 December number of the *Eye, Ear, Nose and Throat Monthly:*

> There is no intracranial lesion which produces dizziness according to the contributors to Alexander and Marburg's latest work on the neurology of the ear. . . . No researcher has ever claimed that vertigo was of central origin. In fact the presence or absence of vertigo has been looked upon as the most deciding factor in the differentiation of central and peripheral lesions in a patient manifesting spontaneous nystagmus.

The following statement is from an article by Brunner [2] in Alexander and Marburg's "Handbuch der Neurologie des Ohres."

> Der Drehschwindel ist vielmehr zunächst charakteristisch für Erkrankungen des Labyrinthes, des Nervus vestibularis und der centralen labyrinthären Bahnen in der Medulla Oblongata. Bei Erkrankungen dieser Gebiete tritt der Drehschwindel als Herdsymptom in Erscheinung. (Vertigo is primarily characteristic for diseases of the labyrinth, of the vestibular nerve and of the labyrinthine tracts in the medulla oblongata. In diseases in these localities vertigo becomes manifest as a localizing symptom.)

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* Read at a meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, Ann Arbor, Mich., Jan. 17, 1931.

1. McKenzie, G. W.: Eye, Ear, Nose & Throat Monthly **9**:468 (Dec.) 1930.
2. Brunner, H., in Alexander and Marburg: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 1, p. 945.

After having carefully examined about 300 patients suffering from injuries to the head, I can state positively that true turning vertigo accompanied by spontaneous nystagmus is present in a fairly considerable number of cases, in which the most thorough and exacting examination of the peripheral vestibular mechanism revealed it to be functioning in a normal manner. It is therefore my contention that in these persons the vertigo is caused by damage to the central vestibular area situated beneath the floor of the fourth ventricle.

Frequently one also finds true turning vertigo exhibited in cases of multiple sclerosis with intact peripheral vestibular end-organs, and in these persons one must assume that the vertigo is caused by an invasion of the central vestibular area by the formation of plaques.

I repeat, therefore, that turning or rotatory vertigo is the most certain sign of vestibular invasion, peripheral or central. There is nothing else in the brain that can produce this phenomenon. The patient suffering from turning vertigo states "that external objects move in a circle about him" or "that he seems to turn in a circle in relation to external objects." As a rule, the movement of the external objects is more frequently complained of, and this movement is always in a horizontal plane. It does not particularly matter whether the external objects or the patient himself moves; the essential fact is that something moves and moves in a circle.

If the patient exhibits true turning vertigo, he must have or must at some time have had spontaneous vestibular nystagmus. This vestibular nystagmus will not be present on every examination. One may have to search long and on various occasions for it. If it is not present as a spontaneous nystagmus, it can be produced by manipulating the patient in such a way as to produce vertigo. The head is suddenly bent forward, the patient stoops toward the floor or the head of the patient is grasped by the two hands of the examiner and slowly rocked backward and forward and from side to side to relax the resistance of the muscles of the neck and then it is suddenly tilted backward. At this moment, the patient is asked to look rapidly to the right and to the left, and a spontaneous nystagmus is elicited if a pathologic process is present. This maneuver is known as Brunner's head movements test. A nystagmus associated with vertigo induced by the movements of the head now appears.

The vertigo seen in a purulent invasion of, or a total exclusion of, the labyrinth by a transverse fracture of the pyramid is violent and is continuous. It begins to improve, however, after a few days, and entirely disappears after a lapse of from five to six weeks, because after this length of time the other labyrinth has learned to compensate for the loss of its mate. On the other hand, the vertigo that is caused by a vasomotor disturbance in the central vestibular area is not continuous,

nor is it so severe as to be completely disabling. It comes on in short spells of a few minutes' duration which are brought on by such factors as rolling over or turning in bed, sitting up suddenly, looking up suddenly, bending the head forward suddenly, going above ground level, undue exposure to sunlight, mental and physical exertion and even the slightest use of alcohol in any form.

The tactile vertigo of Purkinje is not as well known as the turning vertigo and cannot be as surely ascribed to disturbances and diseases of the vestibular mechanism. This type of vertigo is due to disturbances in the muscle sense of the body, and as all of the body musculature is innervated by the vestibular mechanism, it may at times also be due to diseases of this mechanism.

By reason of these errors of sensation, the patient feels as though the bed were tilting or the foot of the bed were rising, as if the ceiling were coming down or as if the side of his chair were falling. He feels uncertain on his feet, as though he were drunk. He complains of things going black before his eyes, etc.

While these errors of sensation are not as important as the true turning vertigo, they must not be considered as hysterical. At times they may be definitely designated as of vestibular origin, but as a rule they are not accompanied by nystagmus. According to some authors, they may represent an earlier stage of turning vertigo.

In chronic adhesive processes of the ears, these errors are found more frequently than turning vertigo, and according to Brunner this is also true in disseminated sclerosis and in cerebellar tumors.

Lateropulsion is another form of tactile vertigo which, in my experience, is not frequently encountered. It was described by Nageotte and Babinski. The patient feels as though he were pushed or pulled toward one side. The pull or push is never forward or backward. The attacks may be very slight or they may be so severe as to simulate epilepsy. These attacks are most frequently encountered in nonsuppurative lesions of the central vestibular area. They are not found in suppurative lesions of the ears. This condition must be differentiated from epilepsy and from hysteria. In hysteria, the patient may fall to one side, but he never says that he feels pulled or pushed to the side. In epilepsy, there is always a loss of consciousness. The patient with lateropulsion never loses consciousness.

It has been stated that turning vertigo is a focal symptom of involvement of the peripheral or central vestibular system and that no other lesion in the brain can produce it. It may be necessary to modify this statement with regard to cerebellar lesions. At present the question as to whether the vertigo associated with cerebellar lesions is an actual cerebellar symptom or whether it represents a pressure or reflex action

on the vestibular nuclei in the pons cannot be answered definitely. The occurrence of turning vertigo as a focal symptom in lesions of the cerebrum is highly improbable.

The duration of the vertigo may assist one to some extent in making a diagnosis. In general, it may be stated that if the vertigo lasts only a short time and then vanishes completely it is more probably being caused by some acute condition of the labyrinth or the vestibular nerve than by a lesion in the brain stem. On the other hand, if the vertigo lasts longer and comes on in attacks over a period of years, it is more apt to be due to some lesion of the central nervous system, although a chronic disease of the labyrinth cannot be excluded. In a peripheral lesion, there is usually an association in the comparative intensity of the vertigo and the nystagmus. When the lesion is central, the vertigo may be severe and the nystagmus mild, or vice versa.

It would be wonderful for the diagnosis of a case if one could assert positively that turning vertigo accompanied by nystagmus is characteristic only of the organic lesions of the peripheral and central vestibular areas, but such is not the case. Any condition that produces vascular changes in these territories can also produce turning vertigo. Among these conditions must be mentioned neurasthenia, hysteria, epilepsy, migraine, cardiac diseases, vascular diseases and the intoxications. In all of these conditions, the modus operandi in the production of the vertigo is undoubtedly found in the production of vasomotor changes in the central and peripheral vestibular systems.

PRIMARY TUBERCULOSIS OF THE NASAL MUCOUS MEMBRANE*

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Primary tuberculosis of the nasal mucosa is a relatively rare condition, characterized by the formation of granulomatous masses, usually on the septum or the inferior turbinates, and by symptoms due to obstruction. The condition is apparently more common abroad than in this country, although the small number of cases reported is probably not a true indication of its frequency here. Chamberlin [1] mentioned having seen ten or twelve cases in dispensary practice, the diagnosis of which was proved microscopically; these cases have never been reported, owing to a lack of accurate records. I have been able to review the reports of only fifteen cases (table 1) cited by American authors,[2] whereas, in Germany alone, in 1928, a single author [3] presented an article based on a hundred cases of tuberculosis of the nose, fifty-one of which were described as not presenting evidence of tuberculosis elsewhere in the body and which probably were comparable to the type under consideration in this paper. The infrequency of the condition seems best explained by the fact that the nasal mucosa is highly resistant, and is protected by an outward flowing, slightly bactericidal secretion.

* Submitted for publication, Feb. 20, 1931.

* From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

1. Chamberlin, W. B.: Nasal Tuberculosis, Ann. Otol. Rhin. & Laryng. **31**: 423 (June) 1922.

2. Farlow, J. W.: Two Cases of Nasal Tuberculosis, Tr. Am. Laryng. A. **15**:63, 1894; New York M. J. **58**:525, 1893. Chamberlin, W. B.: Report of Two Cases of Tuberculosis of the Nose, Laryngoscope **21**:873 (July) 1911. Farnsworth, M. A.: A Case of Tuberculosis of the Septum, J. Michigan M. Soc. **23**:27, 1924. Ballenger, W. L.: A Case of Primary Tuberculosis of the Nasal Septum, Ann. Otol. Rhin. & Laryng. **10**:6 (Feb.) 1901. Freer, O. T.: Nasal Tuberculosis, Two Cases, One Involving Right Ethmoid Bone, Recovering After Operation, Tr. Am. Laryng. A. **31**:103, 1909. Harper, James: Tuberculomata of the Nose: Report of Two Cases, J. Laryng. **34**:81 (March) 1919. Kistner, F. B.: Primary Tuberculoma of the Nasal Mucosa, Tr. Am. Laryng. Rhin. & Otol. Soc. **34**:461, 1928. Thrasher, W.: Primary Nasal Tuberculosis with Report of a Case, Lancet-Clinic **98**:167, 1907. Theisen, C. F.: Tuberculosis of the Nose with Report of a Case of Primary Tuberculosis, Albany M. Ann. **19**:156, 1898; Laryngoscope **4**:79 (Feb.) 1898.

3. Wichmann, P.: Die primäre exogene Infektion der Nasenschleimhaut mit Tuberkulose in ihrer immunisatorischen Bedeutung, Dermat. Wchnschr. **87**:1282, 1928.

TABLE 1.—*Cases Reported in the American Literature*

Author	Cases	Age, Years, and Sex	Complaint	Physical Features: Local	Physical Features: General	Treatment	Result	Diagnosis
Farlow [2]	2	23F	Obstruction; discharge	Thickening of septum; tumor of turbinate	Thorax normal; larynx appeared tuberculous	Curettage; lactic acid	Improved	Not proved
		71F	Obstruction	Soft growth on septum	Not stated	Snare and cautery	Not stated	Made on microscopic examination
Theisen [2]	1	36M	Obstruction	Granular mass on septum	Negative	Snare and cautery	Cure of one and one-half years' duration when reported	Organisms of tuberculosis found
Ballenger [2]	1	45F	Not stated	Nodular tumor with ulceration	Not stated	Not stated	Not stated	Positive results of inoculation of guinea-pig
Thrasher [2]	1	15F	Obstruction	Cauliflower mass on septum and turbinate	Not stated	Snare and cautery	Cure of four years' duration when reported	Not stated
Freer [2]	2	20F	Obstruction; discharge	Granular tumor on septum	Not stated	Surgical excision on two occasions	Cure	Organisms of tuberculosis found
		50F	Obstruction	Granular tumor on septum	Not stated	Partial excision	No record	Organisms of tuberculosis found
Chamberlin: Ann. Otol., Rhin. & Laryng. 31: 423, 1922; Laryngoscope 21: 873, 1911	4	26F	Obstruction	Fungus-like mass on turbinate	Healed tuberculous lesions of one apex	Surgical excision	Cure of one years' duration when reported	Organisms of tuberculosis found
		?M	Obstruction	Cauliflower mass on septum	Not stated	Excision	Not stated	Made on microscopic examination
		38F	Obstruction	Granular mass on cartilaginous part of septum	Not stated	Excision	Not stated	Microscopically, "probably tuberculosis"
		?F	Obstruction	Ulcer on either side of septum, with granular mass	Healed lesion of right apex	Surgical excision	Cure of ten months' duration when reported	Organisms of tuberculosis found
Kistner [2]	1	73F	Obstruction	Firm tumor on cartilaginous part of septum	Negative	Surgical excision	Cure	Results of inoculation of guinea-pig
Harper [2]	2	46M	Not stated	Tumor on both sides of septum	Negative	Surgical excision	Cure of five years' duration when reported	Not stated
		47M	Not stated	Tumor on both sides of septum	Negative, later pulmonary	Surgical excision	Cure of four and one-half years' duration when reported	Not stated
Farnsworth [2]	1	?F	Obstruction	Warty tumor on septum	Negative	Excision twice; radium	Cure	Organisms of tuberculosis found

In this connection, Thomson [4] stated that: "The defenses of the nose are so complete that acute tuberculosis hardly ever occurs there." Trauma, particularly that due to picking the nose, seems to be the chief factor in breaking down this resistance. Infection, when it occurs, is probably air-borne by inhalation of dried, infected secretions, although it is possible that the bacillus of tuberculosis may be mechanically introduced.

Instances of infection have been noted at all ages, but are more often found in middle and later life. Most authors have stated that women are more frequently affected. The five cases reported here (table 2) were in women aged from 50 to 65.

TABLE 2.—*Cases from the Mayo Clinic*

Cases	Age, Years, and Sex	Complaint	Physical Features		Treatment	Result	Diagnosis
			Local	General			
1	65F	Growth in nose	Granulomatous mass in right nostril perforating into cheek	Examination of sputum and Wassermann reaction negative	Radium	Cure of four and one-half months' duration when reported	Made on microscopic examination
2	55F	None	Granular mass on septum	Healed lesion in apex of right lung	Biopsy only	No record	Made on microscopic examination
3	58F	Obstruction	Granular masses either side of septum	Healed lesion in apex of left lung	Biopsy and radium	Cure of four years' duration when reported	Made on microscopic examination
4	50F	Sore in nose	Ulcerated tumor on cartilaginous part of septum	Healed lesion in apex of left lung	Biopsy and diathermy	Recurrence; cauterization by physician at home; cure of ten months' duration when reported	Made on microscopic examination
5	65F	Obstruction	Granular mass on both sides of septum	Negative	Biopsy and radium	Improving, still under treatment	Made on microscopic examination

The tuberculoma is usually a granulomatous mass with a mammillated appearance, situated usually on the septum, although at times appearing on the turbinates, or spreading from either of these to adjacent structures. It may be limited to one side of the septum, or it may be seen on both sides. It is interesting to note the observation made by Kistner that the cartilage between opposing masses is not involved. Ulceration, when present, probably is due to secondary infection following trauma to the tumor and may lead to perforation. Microscopically, there is the usual picture of tuberculosis, with epithelioid cells, giant cells and caseation. It is often difficult to demonstrate organisms of tuberculosis; even inoculation of animals may give negative results.

4. Thomson, St. Clair: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton & Company, 1926, pp. 673 and 688.

The symptoms are those of obstruction, which is due not only to the tumor itself, but to associated, marked crusting. The diagnosis of tuberculosis of the nose must rest largely on microscopic examination of excised tissue, although the condition may be suspected from the clinical appearance of the tumor. The chief lesions to be distinguished from tuberculosis are those of syphilis and of a malignant condition. Syphilis can be recognized, as a rule, by its tendency to affect the bony septum, by the history and by a positive Wassermann reaction of the blood. Malignant disease affecting the septum or turbinates only is rare and may present a clinical appearance similar to that of tuberculosis. The microscopic picture, however, is characteristic.

In the past there has been considerable discussion regarding the relationship of lupus, with involvement of the mucous membrane, to primary tuberculosis of the mucous membrane. Some authors have stated that it is possible to distinguish these forms of tuberculosis microscopically. Broders [5] has expressed the opinion that this is not possible. Goeckerman [6] is in agreement with this view, and further, has pointed out that the controversy has largely disappeared with the advent of newer conceptions of histologic pathology. Clinically, a differential diagnosis usually can be made. In lupus of the mucous membrane there is a marked tendency to scarring, with the formation of "apple jelly" nodules in the scarred areas.

Treatment has varied widely; lactic acid, iodine, roentgen rays, radium, Finsen light, heliotherapy, cautery, diathermy and operation have all been used. Surgical procedures are of two types: wide excision, including the cartilage, and excision with the knife or the sharp curet, leaving the cartilage intact. Chamberlin stated that the latter procedure is followed by rapid healing, and that it has the advantage of not leaving a perforation. His observation fits in well with Kistner's statement, previously noted, that the cartilage is not involved. Cautery or diathermy is next in favor, then radium or the roentgen rays.

The prognosis is generally good, although the condition may recur so that two or three treatments may be needed to bring about complete cure.

Five cases of primary nasal tuberculosis have been observed in the Mayo Clinic. The patients were all 50 years of age or more. Three of the five patients had symptoms of obstruction. All had granular masses involving the septum or turbinates, but in only one instance was ulceration noted. In two cases the septum was perforated, in one instance owing to previous treatment with the cautery. In one case treatment was not given; diathermy was used in one case and radium in three

5. Broders, A. C.: Personal communication to the author.

6. Goeckerman, W. H.: Personal communication to the author.

cases. In the case in which treatment was by diathermy, recurrence developed. The region in which the recurrence occurred was further cauterized by the physician in the patient's home community, who has reported that a second recurrence has not taken place after ten months. Two patients treated with radium were free from recurrence when last observed, four and one-half months and four years, respectively, following treatment. The third patient has been treated too recently to allow any statement as to results.

CONCLUSIONS

Five cases of primary nasal tuberculosis are reported which tend to show that the condition is more common in women in the later years of life than in younger women or in men of any age, that it is characterized by the formation of granulomatous masses on the septum and turbinates, that the symptoms are those of obstruction due to tumor and associated crusting, and that radium and diathermy, or the cautery, are efficient methods of treatment.

THE PATHOGENESIS OF OTOSCLEROSIS*

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Slowly progressing deafness is not always identical with otosclerosis. Increased knowledge of pathologic anatomy and more careful differential diagnoses show that otosclerosis does not occur as frequently as many believe. In numerous cases a slowly progressing deafness is due, not to otosclerosis, but to a chronic inflammation of the middle ear, adhesive processes or progressive degeneration of the cochlear nerve. The problem of otosclerosis is still of paramount interest. Otosclerotic deafness is especially characterized by the fact that it develops in otherwise healthy adolescents or young adults. The otosclerotic bony changes are still mysterious, and have therefore received considerable attention from many investigators.

Not every otosclerotic process in bone, however, leads to so-called "clinical otosclerosis." Whether or not the changes produce progressive deafness depends on the localization in the bony labyrinth. The focal lesions occur in selected areas of the bony capsule of the inner ear and most frequently near the oval window, in which case the pathologic process may encroach on the footplate of the stapes and anchor it firmly to the surrounding bone (ankylosis of the stapes). When this occurs, the stapes loses more or less of its ability to oscillate and to transmit sound to the inner ear, and a disturbance in function results. The region near the footplate of the stapes, although the most common situation for foci, is by no means the only place of predilection, the disease of the bone being frequently found also in other regions of the labyrinthine capsule, especially in the capsule of the cochlea and the promontory. In certain severe cases the otosclerotic process may expand so as to involve the entire labyrinthine wall to the region of the semicircular canals.

The anatomic picture of otosclerosis has been known for a long time, many investigators having described it as clearly different from the normal structure of bone. Notwithstanding this no one has fixed the pathogenesis of the process. In the past, the primary cause and the development of the bony changes have been expressed only in the form of various hypotheses. In the first place, the insufficiency of knowledge

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lies in the fact that the disease is peculiar and not identical with pathologic processes in other parts of the skeleton. I am therefore of the opinion that to compare it with other bony processes of the human skeleton is not of value in research into its pathogenesis.

This peculiarity of otosclerosis depends on some special property of the bony structure of the labyrinthine capsule. All investigators agree that the bony labyrinth normally differs from all other bones of the skeleton. It is formed on a cartilaginous capsule by endochondral ossification which starts from so-called points of ossification in contradistinction to other endochondral bones. The labyrinthine capsule attains its final structure and size early in life and normally does not undergo further transformation. Consequently, the distribution of the blood vessels, which is determined by the points of ossification, also remains practically unchanged; the different regions supplied by these blood vessels are more or less separated from one another, and new anastomoses cannot develop between them in later life. These peculiarities of the bony capsule are found not only in man, but in other mammals, as for example in the animals generally used for experimentation (guinea-pig, rabbit), and even in certain nonmammals, i. e., birds. Thus by investigating pathologic processes in the bony labyrinth of these animals one may be able to expedite the interpretation of otosclerosis in man. In animals, however, spontaneous diseases of the labyrinthine capsule similar to otosclerosis have not yet been observed. Nevertheless, for many years, I have found pathologic processes in the labyrinthine capsule of the hen which show a striking similarity to human otosclerosis.

As often occurs in scientific work, I discovered these changes accidentally in studying the effect of the injection of certain substances into the inner ear. In some cases, the experiment was stopped because of a venous bleeding, which was checked by tamponing. In these cases, microscopic sections showed changes in the bone that were astonishingly similar to those in human otosclerosis. I assumed that the changes were caused by venous stasis. With this idea in mind, I systematically performed a great number of experiments, the results of which I give briefly.

RESULTS OF EXPERIMENTS

In my first experiments, published in 1919, I attempted to create a venous stasis in the sinus that accompanies the semicircular canals of the hen. In further experiments, however, the intracranial median sinus proved to be more accessible and satisfactory. This sinus lies in the same place in which the cisterna magna is found in the cat, dog, rabbit, man, and other animals. It is easily punctured. In the first experiments the puncture was repeated three or four times after trephining the skull. In all cases the circulation in the sinus was stopped by the introduction

of ferric chloride into the lumen. By this means sufficient venous stasis was usually produced to bring about the bony changes described. In 80 per cent of the experiments, the positive result was evident, in 10 per cent it was somewhat uncertain, and only in 10 per cent a result was not obtained. If one keeps in mind that the venous system is enormously variable and that the blood may take a vicarious way through newly formed anastomoses, the high percentage of positive results appears significant. The experiments prove without doubt that the resulting changes are due to the artificially produced venous stasis.

The many results obtained by the aforementioned method can be discussed here only in a general way. At the congress of the Collegium Oto-Rhinolaryngologicum Internationale in London in 1929, I gave a more comprehensive review. By means of forty colored photomicrographs, I demonstrated that all pictures of otosclerosis in the human being that have been published might be compared with my analogous pictures experimentally produced. This is noteworthy if one considers that human otosclerosis also occurs in various pictures that may be interpreted as different stages, and also as different degrees of intensity of the pathologic process. In order to explain better the genesis of the pathologic process, I shall describe the initial stage, the high points and processes of a fairly high degree of intensity.

There is an anatomic difference between the bony capsules of man and those of the hen, particularly in regard to the distribution of blood vessels (recognizable by low magnification). One may expect, therefore, differences in the arrangement of the pathologic foci in the bone. However, this fact is of minor importance. The deciding point for coupling the two conditions is without doubt the similarity of the biologic processes that take place in the bony substances. To bring out clearly the similarity between the pictures of otosclerosis and experimental bony changes, it is best to use a high magnification.

Figure 1 shows that there is not a marked difference between the normal bone of the human being and that of the hen. The histologic picture of the normal bony capsule is especially characterized by relatively narrow vascular canals bounded by a narrow zone (staining blue). The great affinity of this zone for hematoxylin is evidently caused by deposits of calcium. In the vicinity of the vascular canals is found a pink-stained bone with typical haversian lamellae. The vascular canals in the labyrinthine bone in the hen are relatively wider and lie nearer to one another than do those of man. However, this cannot be regarded as a major difference.

Figure 2 shows the changes most frequently observed in otosclerosis. Without doubt these correspond to the high point of a process of medium intensity. Figure 2*A* demonstrates otosclerotic changes in man and figure 2*B*, the analogous changes elicited by experiments on the hen.

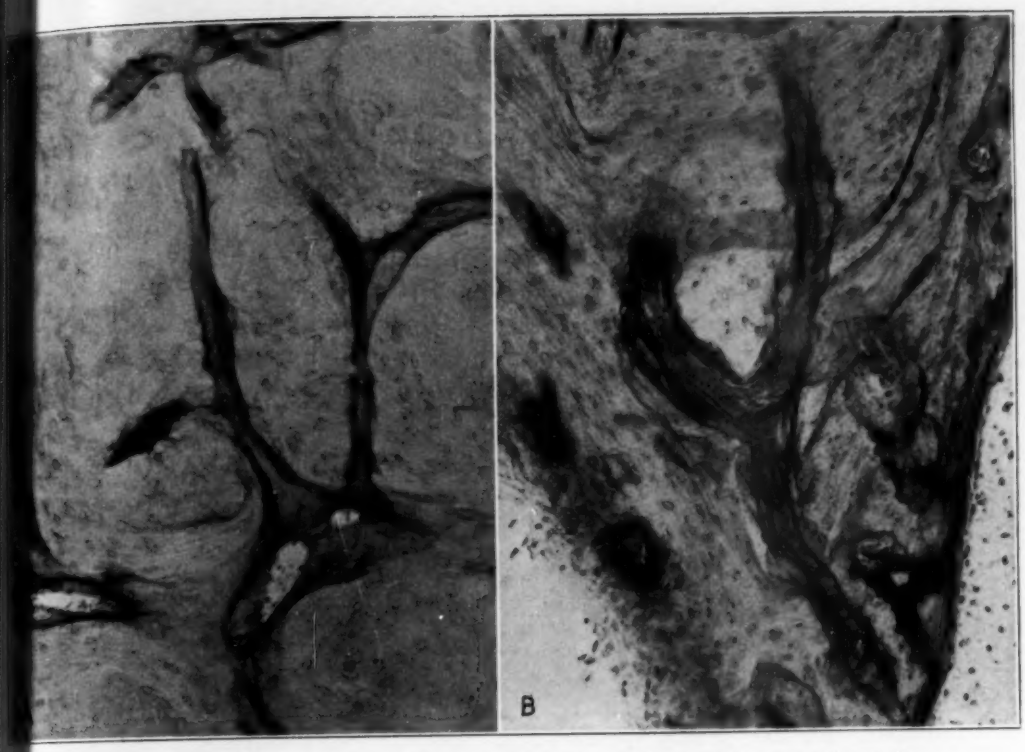

Fig. 1.—Photomicrograph of the normal bony capsule of the human being (*A*) and of the hen (*B*).

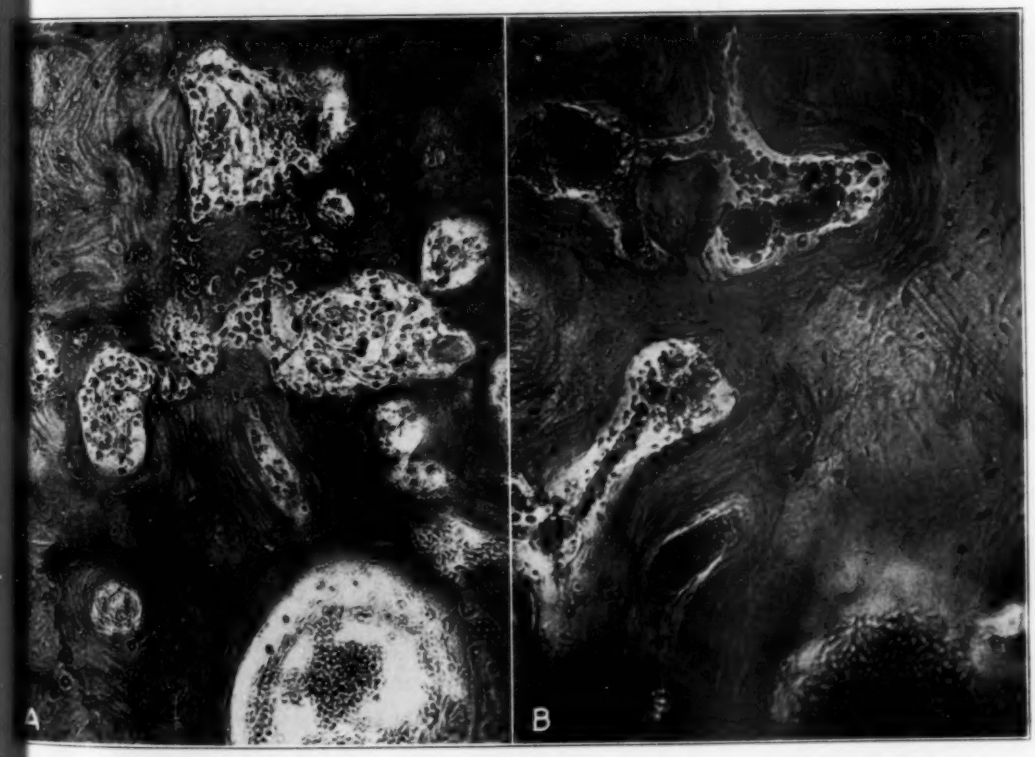

Fig. 2.—The changes most frequently seen in otosclerosis. *A* demonstrates the otosclerotic changes in man, and *B*, the analogous changes produced experimentally in the hen.

Both conditions reveal the same bony changes that are generally recognized as typical of the otosclerotic process. The vessels are unusually large and full of blood in comparison with the normal state. The enlargement takes place exclusively in the thin-walled veins of the bone. A zone is found around the enlarged vessels within which the bony substance is absorbed and its place taken by a tissue moderately rich in cells. The calcified zone (staining deep blue) that normally encircles the vascular canals in a narrow line is lacking. Instead, for a consider-

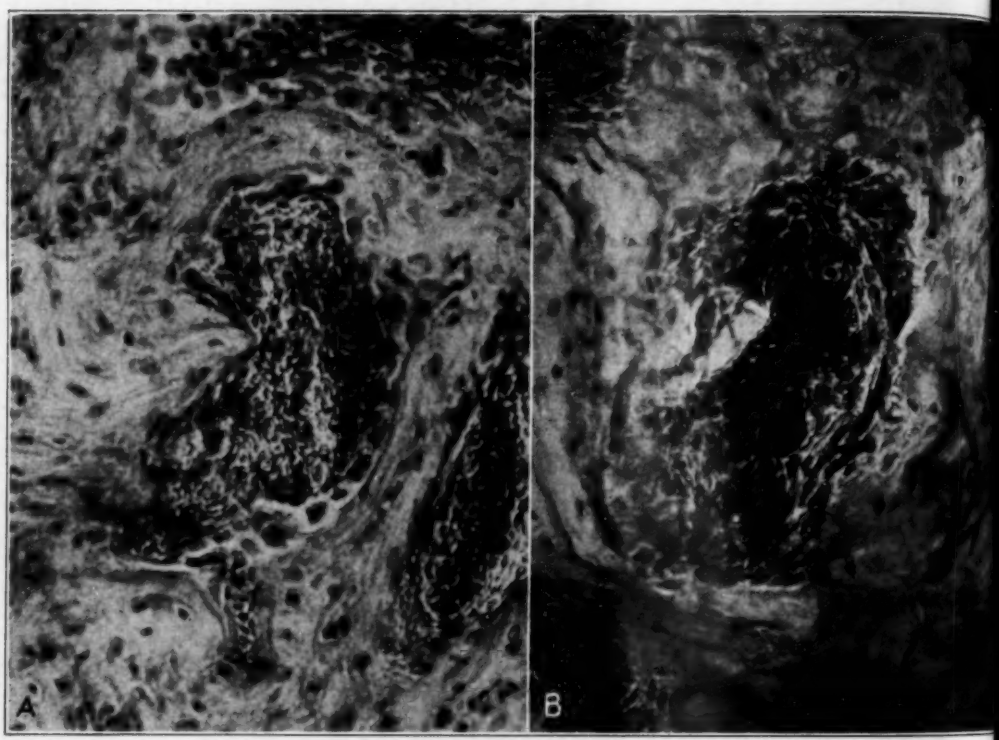

Fig. 3.—In this figure the vessels are larger and are engorged with blood to a greater extent than those shown in figure 2. The specimen in *A* was taken from a case of human otosclerosis; *B* reveals analogous changes produced by experiment.

able distance around each vessel the bony substance is stained more homogeneously deep blue than under normal conditions. Within this blue-stained region are found an obvious increase and enlargement of the cavities and cells of the bone, the so-called sieve holes, "siebförmige Durchlöcherung" (Manasse). These changes are observed with striking uniformity in the circumference of all the vessels within the region of the otosclerotic focus.

In the conditions shown in figure 3 the vessels are larger than in figure 2 and are engorged with blood to a much greater extent. Figure 3*A*, taken from a case of human otosclerosis, shows a similar condition. I have observed this stage only once before (1919). Figure 3*B* reveals analogous changes produced by experiment. I regard these conditions as initial stages of otosclerosis caused by a process of maximum intensity. This picture suggests the well known pathologic appearance of organs in which stasis of the blood has occurred (in the lungs, liver and kidney). The enormous enlargement and engorgement of vessels can be explained only by a stasis in the venous circulation. Why should it be assumed, therefore, that some other condition could produce the same anatomic picture in bone (fig. 3)? Is it not evident that vascular stasis is the primary causative factor? Moreover, the aforementioned experimental stasis produces the identical picture. The initial stages, therefore, show only narrow zones of resorption around the enlarged vessels. These zones are completely filled with large cells resembling osteoblasts (and occasional others resembling osteoclasts) evidently derived from a proliferation and transformation of osteocytes. The pictures shown in figure 4 evidently developed during a later period of the disease. The specimen in figure 4*A* was taken from a case of clinical otosclerosis. Figure 4*B* represents the corresponding condition produced experimentally in the hen. In these pictures the vessels are still obviously enlarged, but not as much as in the foregoing stages (fig. 3). A zone of resorption has developed around the vessels, which, in comparison with figure 2, is still more extensive and contains more cells. Under high magnification, the remaining bony regions reveal the same changes as the bone in figure 2; that is, they stain a deeper blue and show the sieve holes "siebförmige Durchlöcherung."

This stage of the otosclerotic process, both in the human being and in experimental animals, bears a distinct similarity to a certain stage in the process of normal endochondral ossification. To confuse these two morphologic pictures as O. Mayer asserted that I have done is impossible, because only adult hens with fully developed bony capsules have been used in all my experiments. In old hens, such pictures never occur as normal peculiarities. A more detailed study clearly reveals differences between pathologic changes and developmental stages, especially in regard to the blood vessels. Moreover, in some experiments otosclerotic changes occur in the form of a circumscribed focus sharply set off from the intact and normal regions of the labyrinthine bony capsule, as is the case in otosclerosis.

The high point of the process in both medium intensity (fig. 2) and higher intensity (fig. 4) can evidently be delayed for a relatively long time. Sooner or later, however, a stage follows that is characterized

by an obvious decrease of the stasis and a gradual increase of the reossification. The resorption cavity around the vessels gradually refills with the newly formed bony tissue and thus diminishes in size. However, the newly formed bone resulting from the otosclerotic process in the human being or the animal clearly differs from the original bone of the normal labyrinthine capsule. The vessels and vascular canals in the newly formed bone remain a little wider and are more full of

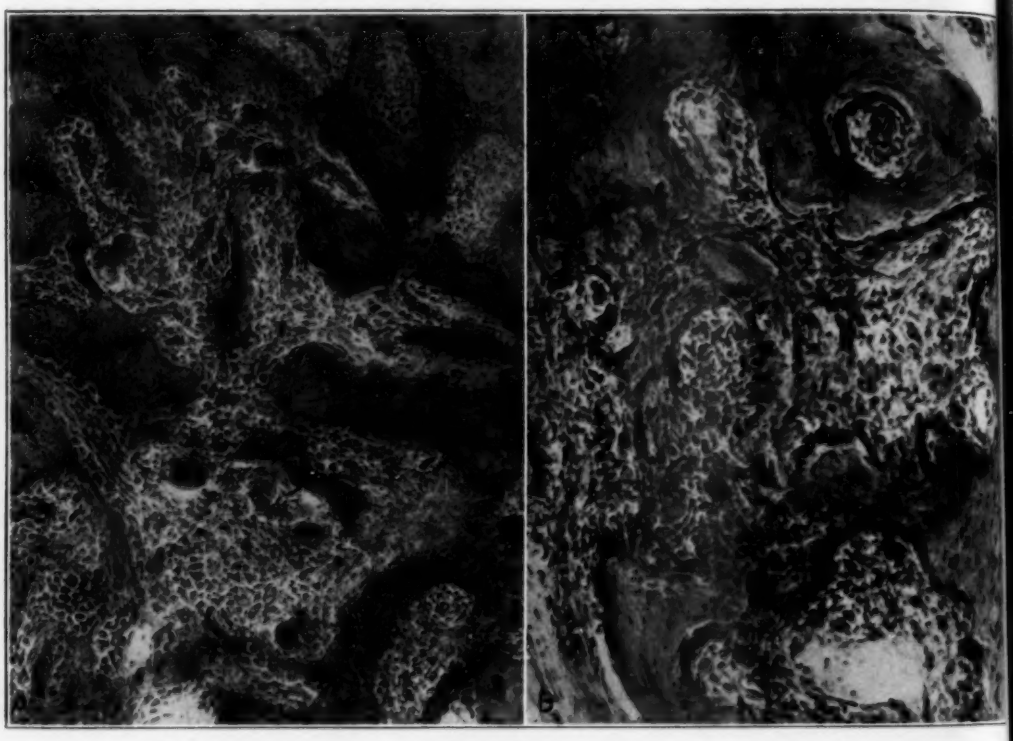

Fig. 4.—Photomicrograph of a specimen in a later stage of the disease. The specimen in *A* was taken from a case of clinical otosclerosis; *B* represents an analogous condition produced experimentally in the hen.

blood. In the transformed bone of an otosclerotic focus, the narrow halo that stained a deep blue around the vessels is lacking. The bony substance is not laid down in the form of regular haversian lamellae, but shows a more irregular structure. Moreover, the substance is stained a brighter pink than normal bone. The similarity between the picture in the human being and that in the experimental animal is evident from the accompanying illustrations.

CONCLUSIONS

I shall now discuss the conclusions that may be drawn from the experimental observations on the pathogenesis of otosclerosis. The striking similarity of the experimentally elicited conditions to those found in human ostosclerosis leads to the conclusion that the bony changes in man may be traced back to the same or, at least, a similar cause. There is no doubt that in the experiments described a stasis within the veins of the bone was produced, and that the bony changes obtained by this method were due to the venous stasis. In consequence, it must be assumed that human otosclerosis is caused by similar influences which have led to a stasis of blood within the veins of the bone. The pathologic process may then be regarded as caused by the stasis or by the disturbance of the normal relation of hydrogen and hydroxyl ions within the tissue closely contiguous to the vessels involved. No doubt, as a consequence of the stasis, the quantity of the H-ions increases within these regions of the bony tissue, and it appears that the bony substance responds by a typical biologic reaction. The manifestations of the process in the culminative stage may be interpreted as consequences of "halisteresis." Therefore, the conclusion was reached that pathologic process of otosclerosis in the labyrinthine capsule is caused by a venous stasis within the veins of a circumscribed region of bone. Space does not permit a comprehensive discussion of the different objections raised against this view. I have confined myself to a statement of the experimental facts and the general principles of the theory.

Similar reactions have not been observed in other bones of the skeleton. The fact that they occur exclusively in the labyrinthine capsule depends, as already stated, on the special property of the endochondral ossified layer of the labyrinthine bone and the special distribution of blood vessels. This distribution evidently contributes to an eventual stasis of the blood within the veins of the capsule of the bone which cannot be relieved by anastomoses with vessels in adjacent regions. In consequence, an extraordinary pressure must arise within the veins of the bone, which evokes the otosclerotic reaction.

In spite of this knowledge, the problem of otosclerosis is not solved. The question now arises as to the cause of the venous stasis. For the time being, I cannot answer this question definitely. A single cause does not suffice to prove my contention. In the experiments on the hen, stasis was produced by traumatic stricture of the venous sinuses. One must not conclude, however, that the same condition is responsible for the genesis of human otosclerosis. Certainly a traumatic stricture in the venous circulation of man can seldom be regarded as the cause of otosclerosis, as indicated by the repeatedly reported, but still much contested, cases of traumatically caused otosclerosis. In the preponderant

majority of cases, the otosclerosis is certainly due to other factors. To determine these factors requires further research.

Moreover, attention may be called to the fact that this theory of pathogenesis of the otosclerotic process does not conflict with the assumption of a hereditary disposition, which several authors have shown to exist. However, the hereditary factor is not observed in all cases of otosclerosis, because fairly often one may find cases in which such a disposition is not traceable in spite of thorough genealogical inquiries. This fact also contributes to the assumption that a number of different factors may be responsible for the otosclerotic process. The cases of hereditary otosclerosis may easily be brought into accordance with the theory of venous stasis. The reaction of the vessels, as already described, in all probability is facilitated by an inherited inferior functional quality in the vascular system, especially within the walls of the vessels. In a similar way, the genesis of varices, for example, the formation of varicose veins in the leg, may be explained. This well known process of venous stasis cannot be traced exclusively to a purely mechanical cause derivable from habits or similar factors. No doubt, also, in the genesis of these changes special individual hereditary dispositions leading to an early dysfunction of the vascular walls must play an important rôle.

In regard to the otosclerotic focus that is situated near the oval window and that may produce clinical otosclerosis, an analogous factor, as in the formation of varices, may be taken into consideration. As a rule, the chief vein that leaves this focus is clearly recognizable in microscopic sections and may be relatively easily followed in its further course. For a considerable distance this vein accompanies the nervus petrosus superficialis minor and is embedded in rigid-walled surroundings. Moreover, after leaving the skull the vein evidently does not directly join one of the chief branches of the jugular vein, but first runs into the plexus pterygoideus. It is conceivable that because of these anatomic peculiarities a circumscribed disturbance of the circulation of the venous blood that comes from the affected region of the labyrinthine capsule may possibly develop.

The foregoing deductions show that in all probability the pathogenesis of otosclerosis may be explained by the theory that I propounded in 1919.[1] This theory is based on noteworthy facts and especially on experiments that may be repeated for verification. Other hypotheses that cannot be gone into in this paper are built only on speculative reflections.

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1. Wittmaack, K.: Die Otosklerose auf Grund eigener Forschungen, Jena, Gustav Fischer, 1919.

MELANIN IN THE INNER EAR*

DOROTHY WOLFF, M.A.
ST. LOUIS

REVIEW OF LITERATURE

The presence and distribution of melanin pigment in the labyrinth has long been recognized. In recent years, however, this fact has been overlooked by the authors of general textbooks of histology in their discussions of both pigment and the cytologic structures of the inner ear. English textbooks, in particular, have been remiss in this respect.

In 1851, Corti described pigment cells, resembling those of the choroid coat of the eye, in the "periosteum" (spiral ligament) of the cow and sheep. He observed pigment in amorphous grains and in enlarged epithelial cells of the stria in "very old" cats. It was also noted along the aqueduct of the cochlea and, in the cow and sheep, along the sheath of the auditory nerve.

As early as 1860, Voltolini of Breslau, in the first of a series of three reports on postmortem conditions of human ears, cited the presence of pigment in the cochlear spindle of a tuberculous patient aged 35. In the same article is the record of a tuberculous patient aged 37, in which it is definitely stated that no pigment was present. Pigment is not mentioned in the other three cases of the report. In his second article (1861), Voltolini reported four cases, in one of which a boy aged 16 showed a great amount of pigment in the three canals. In a man aged 65 with sarcoma in the internal auditory meatus, pigment was not mentioned. In 1863, Voltolini's third article appeared. In this he gave a description of fourteen cases, three of which showed pigment. In one of these patients, a man aged 81, pigment was found on the secondary tympanic membrane and most heavily on the spindle. A man aged 86 (whose death was due to heart failure and Pleuraerguss [pleural effusion]) showed pigment in the canals. Pigment was found in the saccule of a third patient whose age was not given and whose death was due to typhus.

In 1864, Lucae presented seven cases in a report similar to that of Voltolini, four of which showed pigment in the labyrinth. In a man aged 42, who died of tuberculosis, he observed much pigment where the nerve fibers spread out to the saccule and the ampullae, also along the cochlear spindle and Corti's membrane. He stated that the

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* From the Departments of Pathology and Otolaryngology of Washington University.

processes of these cells anastomosed with each other. In a man aged 47 with Bright's disease he observed round clumps of pigment in the spaces between Corti's fibers, and star-shaped branching cells on the wall of the saccule. Pigment was also observed in a man aged 70, who died of an infection in the knee joint, and in a man 50 years of age with tabes dorsalis. Lucae stated that the pigment might not be very important, as he had observed it in the rabbit. He did not think that it had a pathologic significance.

Von Rüdinger, in 1871, gave many illustrations of pigment cells in the vestibule and canals of amphibia. In discussing the human labyrinth, he said that Henle considered that the periosteum corresponded to the subarachnoid, and that perhaps that is why pigment cells are so scarce.

Waldeyer (1871) saw a strong connection between Corti's organ and the retina. He found the connective tissue walls or the ductus cochlearis and the choroid coat comparable, and he called attention to the similarity of the pigment cells in the ear and the eye.

In his detailed monograph, Retzius (1884) illustrated pigment cells and stated that he observed them in the human being on the vestibular membrane (Reissner's membrane). Reviewing Hasse's work on birds, he stated that pigment cells are lacking.

In 1901, Alexander made a detailed comparative study of the presence of pigment in many mammals. He found it present in most mammals in varying amounts. In one cat it was not found. (Unfortunately, the author does not state the color of the animals observed. One wonders whether the cat was the white cat that he has described elsewhere.) Pigment is found in the axial connective tissues of the cochlea in the human being, and in the endosteum of the scalae in the new-born infant and frequently in the adult. He found it also in the stria vascularis and in the connective tissue of the spiral ligament in the secondary tympanic membrane, in the vestibular floor, at the bases of the cristae maculae utriculi and sacculi and in the membrana vestibularis. He considered the pigment cells morphologically like those of the choroid coat. He presented excellent illustrations in color, showing pigment in various characteristic places in the labyrinths of various animals.

Siebenmann, in describing Reissner's membrane, stated that spindle-shaped cells with or without pigment had been observed. In describing the spiral sulcus, he observed that the epithelium is frequently pigmented. In the cochlea, suspended along the modiolus connective tissue, he noted cells of embryonic type resembling the arachnoid and bearing pigment.

Ranvier (1889) described pigment cells in the stria vascularis of the guinea-pig. He considered that they represent in the cochlear

canal the homologue of the epithelial pigment of the retina; he presented a diagram to illustrate his point. He did not mention their presence in the vestibule.

Dawson (1925), in a comprehensive study of pigment in melanomas, failed to mention the presence of pigment in the labyrinth when discussing the general distribution of pigment in the body.

Few of the recent general textbooks on histology or pathology mention the inner ear in the list of pigment-containing organs of the body, such as the eye, the skin, the arachnoid of the brain and spinal cord, etc. Shambaugh (1927) made no mention of the presence of pigment in the labyrinth. Most of his illustrations seemed to be from other than human material. If a white animal was used it is probable that no pigment was present.

Jordan (1924) failed to mention the presence of pigment in the labyrinth. Lewis and Stöhr (1927) did not refer to it, nor did Bohm, Davidhoff and Huber (1920).

The most complete and accurate statements were made by W. Kolmer (1927). He stated that the amount of pigmentation in the region of the labyrinth depends on the general pigmentation of the animal. It is lacking in albino animals. In 1923, Eisinger found pigment in the sensory epithelium of the crista of the elephant. In the human being it is found especially in the modiolus, and increases with age. The cell bodies range in size from ⅓ to 3 microns. According to Alexander, pigmentation may be found in the bony vestibular canals, membrana tympani secundaria, epithelial wall of the sacculus, endosteal vessels and Reissner's membrane.

Maximow (1930) stated that branched melanophores are often encountered in the tissue of the utricle and saccule. The connective tissue of the spiral ligament also contains stellate connective tissue cells filled with pigment and other inclusions. The vestibular membrane sometimes contains pigment cells.

Even less informing are the textbooks devoted solely to the anatomy and diseases of the ear. Kerrison (1930) did not mention the existence of pigment in the labyrinth. Politzer (1926) briefly mentioned it and referred to Alexander's work. Henke and Lubarsch (1926) did not index the subject of pigment, but in the chapter by A. Eckert-Möbius the presence of pigment is discussed and the contributions of Rüdinger, Retzius and Alexander are mentioned. A. A. Gray (1907) observed that pigment is absent in human labyrinths, in many examples at any rate, and that it would be interesting to know whether pigment is found more abundantly in the labyrinth of the Negro than in that of white races; he said that probably no such investigation had ever been carried out.

OBSERVATIONS FROM THE OTOLOGY LABORATORY OF WASHINGTON UNIVERSITY

The observations thus far accumulated in the otology laboratory of Washington University are presented in order to call attention to this neglected subject. The summation of data from various investigators may lead to significant conclusions in the future.

The membranous labyrinths of twenty-eight human beings were examined microscopically, from sections cut 20 microns thick (with the exception of five embryos sectioned at 10 microns). Observations were also made on the monkey, rat, cat and rabbit. The race of the five embryos and one infant in the collection is unknown. The human specimens examined were from three 2 month embryos; three fetuses from 3 to 6 months old, two premature infants (6 months and 7 months, respectively), two stillborn infants and seventeen other persons rang-

TABLE 1.—*Findings in Embryos and Fetuses*

Autopsy No.	Crown-Rump Length	Approximate Age	Pigment in		
			Eye	Ear	Skin
XVI	7.2 cm.	4 months	+	—	—
XVII	15.15 cm.	6 months	?	—	—
36	15 mm.	2 months	+	A few red-staining inclusions, shaped somewhat like pigment cells	—
37	21 mm.	3 months	+	—	—
49	20 mm.	2+ months	+	—	—
124	17 mm.	2 months	+	—	—

ing from 19 days to 59 years in age. The first table shows the findings in the embryos and fetuses. Table 2 shows the findings in the older specimens.

The observations in table 1 would indicate that pigment in the eye appears earlier than at 4 months, as stated by Spencer. Observations were made on the skin in these cases in view of the statement of Dawson that in the fair races all basal cells are pigmented at birth and that in the fetus all the basal cells are pigmented for a long time; later a depigmentation occurs. In the present specimens it will be noted that no pigment was found in the skin, even in a 6 months premature Negro infant that lived one day.

Of the thirteen specimens showing pigment, five were from Negroes and eight from white persons. It is interesting to note that all the Negroes examined showed a heavy deposit of pigment, with the exception of one 6 months premature infant which gave negative results. Pigment had probably not yet formed.

Those showing pigment ranged from two stillborn infants to persons 59 years of age. Of the white persons not showing pigment none was

older than 10 years. It is conceivable that if it could be applied, the "dopa" reaction might exhibit pigment in these apparently negative specimens.

The relation of the deposit to sex is as follows: In males, eight of eleven showed pigment; in females, five of ten. Of those who did not show pigment two were males and five females.

In all cases in which both ears were examined, the condition—either positive or negative—was found to be bilateral.

TABLE 2.—*Findings in Older Specimens*

Autopsy No.	Pigment	Fixative*	Race	Sex	Age	Diagnosis
I	—	Form.	?	?	?	Congenital syphilis
XVI	—	Z-F	W	F	7 months (premature)	
3849	—	Form.	W	M	19 days	Congenital atresia of the esophagus
3865	+	Form.	W	M	4 months	Congenital syphilis; otitis media; thrush; spleen the size of an adult's (10.5 by 0.5 by 1 cm.); weight, 60 Gm.; pulp soft
3869	—	Form.	W	F	11 weeks	Spina bifida; operation for meningocele
3873	+	Form.	W	F	3½ months	Purulent bronchitis; splenomegaly; bronchopneumonia; congenital syphilis
3878	—	Form.	W	F	15 months	Peritonitis; bronchopneumonia; edema of the brain
3884	+	Z-F	W	M	36 years	Multiple areas of cerebral softening; several arachnoid cysts found at operation
3887	—	Zenk.	N	M	1 day (6 months premature)	Prematurity; atelectasis; hemorrhage beneath skin; congenital malformation of the brain
3894	+	Z-F	W	M	51 years	Spongioblastoma multiforme
3903	+	Form.	W	M	8 years	Fracture of skull
3920	+	Z-F	W	F	5½ months	Hydrocephalus (acquired)
3923	+	Form.	N	M	10½ months	Sympatheticoblastoma of suprarenal; metastases to skull, ribs and mediastinal lymph nodes
3924	+	Form.	N	M	Stillborn	
3937	+	Zenk.	W	F	Stillborn	Icterus; massive hemorrhage in gastro-intestinal tract
3947	+	Zenk.	N	F	7 months	Influenzal meningitis
3948	+	Form.	N	M	13 years	Ewing's sarcoma of the right fibula; metastasis to the skull, vertebrae and ribs
3951	—	Zenk.	W	F	5 years	Streptococcus meningitis
3953	+	Zenk.	W	M	59 years	Carcinoma of larynx
3956	—	Zenk.	W	F	10 years	Lateral sinus thrombosis from mastoiditis
3967	—	Form.	W	M	2 months	Congenital hydrocephalus
4106	+	Form.	N	F	1 year	Tuberculous meningitis

* Form., formaldehyde; Zenk., Zenker's solution; Z-F, both solutions.

In the belief that some correlation might exist between the presence of pigment in the labyrinth and its occurrence elsewhere in the body, an examination was made of sections taken from other regions. Sections of the brain, skin, suprarenal gland and spleen were sought in particular. The results do not merit report, as the tissues were not taken uniformly, no such problem being in mind at the time of autopsy. It may be said, however, that pigment was found in the arachnoid over the brain stem when no pigment had been reported in the cochlea, and vice versa.

The animals studied included five monkeys, two albino rats, one rabbit and three cats. The monkeys were all of the species *Macacus rhesus* and were sectioned at 20 microns. In all of these, pigment was observed. No pigment was found in the albino rats, as was to be expected. Of the cats, two were late fetuses and showed no pigment. The third was an adult cat, almost entirely black. One ear was examined by the dissection method which proved rather unsatisfactory for the exact location of the pigment found. The other ear will be prepared for serial sections. Material for observations is being collected from blue-eyed white rabbits and guinea-pigs.

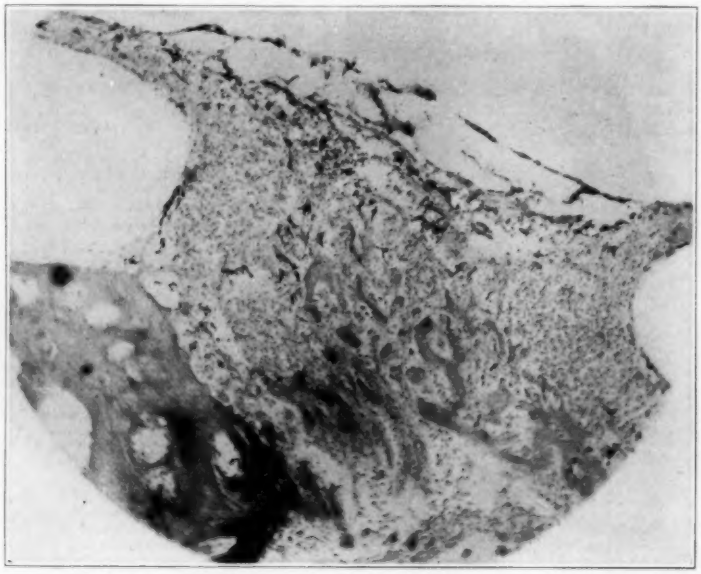

Fig. 1 (autopsy 3894).—Distribution of pigment on the modiolus; × 50.

DISTRIBUTION OF PIGMENT IN THE INNER EAR

In the human material under consideration, the modiolus is found to show more abundant pigment than any other region. If pigment occurs at all, it is always present on the modiolus. Here it lies in the delicate connective tissue cells in the interstices of the cribriform area and along the connective tissue forming the proximal wall of the scalae. The amount is generally greater along the modiolar wall of the scala vestibuli than along the modiolar wall of the scala tympani. If the amount present is slight, it will be found along the scala vestibuli and not along the scala tympani. If the amount of pigmentation is very small, the chromatophores may be seen only along the bony wall instead of in the membranous meshwork, and appear to accompanv

the modiolar artery. When less dense, the pigment appears as fine granules within the primitive branched connective tissue cells.

In cases of heavy pigmentation the melanin may be seen along the lamina spiralis ossea, almost to the labium spiralis. On these delicate bony trabeculae, it appears to cling to the periosteum. It never occurs among the spiral ganglion cells and rarely close to the nerve fibers. In the specimens examined, it was not found (as reported by earlier observers) in typical bone marrow spaces, but only in the interstices in the bone through which nerve fibers pass, or in those filled with delicate areolar tissue, such as is commonly seen in sections of the temporal bone. In only one specimen, in one place, did the pigment appear on

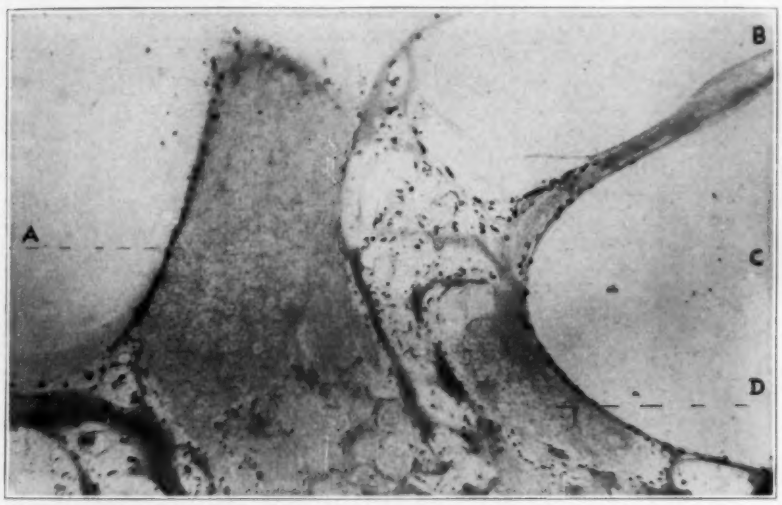

Fig. 2 (autopsy no. 3923).—Photomicrograph showing how the deposition of pigment avoids the region of the ganglion cells. *A* indicates ganglion; *B*, scala vestibuli; *C*, scala tympani, and *D*, ganglion; × 50.

the bone. This was probably a surface cut of an interface of the bone, over which a branch of a melanophore happened to pass.

Besides being found in the cochlea, pigment was also observed in the interstices of bone through which the branches of the vestibular nerve pass to their terminations. It frequently occurs at the base of the maculae utriculi and sacculi and at the bases of the cristae in the characteristic manner so well illustrated by Alexander. In two persons, one Negro and one white, it was observed in the round window membrane.

Sometimes pigment was observed at the bases of the ampullae of the superior and horizontal canals and not at that of the posterior canal. Sometimes the pigmentation of the horizontal canal appeared heavier

than that of the superior canal. Perhaps these differences may be related to the route of the blood supply.

Pigment also occurred along the wall of the cisterna perilymphatica, and in one case it was observed along the wall of the endolymphatic duct. In only one case was it seen on Reissner's membrane. This is significant, as its occurrence here and in the stria would seem to be rather common, according to some authors. Perhaps the fact that the delicate Reissner's membrane was cut in cross-section in the present

Fig. 3.—Individual pigment cells as seen under high power.

specimens and never seen in surface view makes these chromatophores less readily observed. Actual large branching chromatophores were not found in the spiral ligament, though granular pigment was observed, especially along the stria vascularis.

Pigment was not found along the facial nerve or along the eighth nerve in the internal auditory meatus.

The distribution of pigment in animals seems to differ in certain respects from that in man. In none of the lower mammals examined was it ever observed in the delicate connective tissue within the interstices of the cribriform modiolus. (A marked anatomic difference between the monkey and man was noted in this region. The nerve fibers

in the monkey completely fill the canals in which they travel, leaving no space for loose areolar tissue to surround them. The interstices through which the cochlear blood vessels pass, however, are well surrounded by the perivascular connective tissue, but even in this region no pigment was observed.) Pigment was not found in the lower mammals along the medial wall of either the scala tympani or the scala vestibuli. In all animals in which it was found, it occurred along the stria vascularis. In the monkey it could be observed only under high power

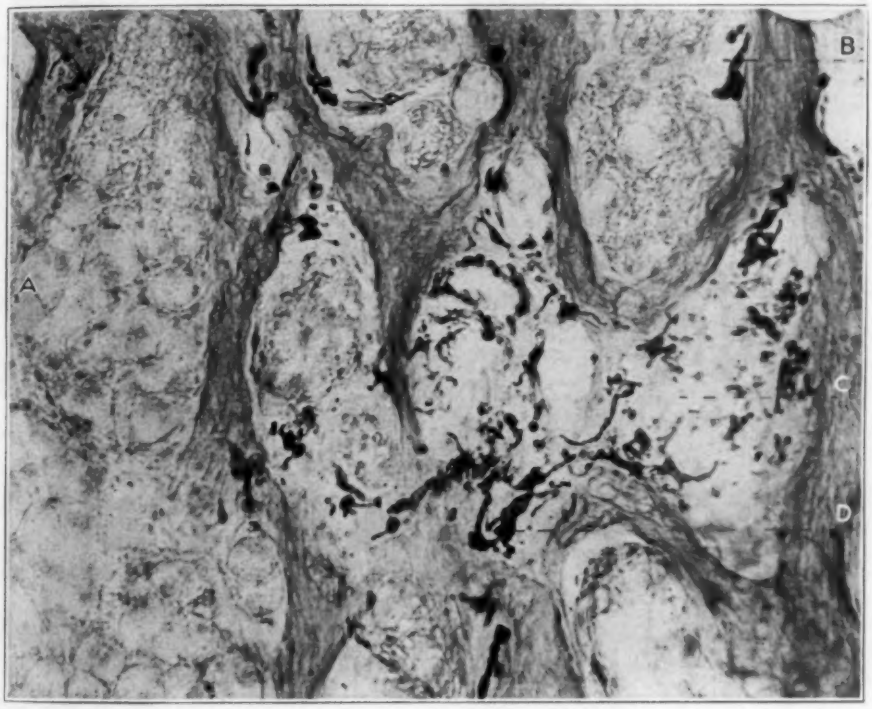

Fig. 4 (autopsy 3948).—Distribution of pigment in perivascular and perineural spaces of the modiolus. *A* indicates cochlear neurons; *B*, perineural space; *C*, perivascular space, and *D*, pigment.

magnification. In one monkey it was found dispersed in fine granules along Reissner's membrane. (It was not observed in Reissner's membrane even in the heavily pigmented specimens from human beings.) It was observed in the perilymphatic tissue below the level of the cristae of the horizontal and superior ampullae.

The pigment observed in the rabbit was relatively heavier than that observed in the monkey, although the animal was a mixed white and New Zealand red.

THE CHARACTER OF PIGMENT CELLS

Human specimens showing an abundance of pigment exhibit large, irregularly branched chromatophores the "antler-like" processes (Loeb) of which seem to anastomose with each other. They are easily discernible under low power magnification. It is readily conceivable that they may even form a continuous network similar to that which is thought to exist in the subcutaneous pigment of amphibia. The thick celloidin sections (20 microns) make the study of these chromatophores

Fig. 5.—Pigment at the point of attachment of the round window membrane.

much more informing than thinner sections. By focusing, it is possible to trace the ramifications of the processes. These twist in more or less regular spirals, as one may see by following the parallel rows of granules. The branching in the arachnoid of the brain is not nearly so evident in the specimens examined, even though some sections were also cut at 20 microns.

In places the chromatophores may appear as pale yellow branched cells, slightly granular. They frequently appear to have grown like an amorphous crystal. Perhaps they can better be likened to the budded branches seen on a copper sulphate crystal when it is immersed in

a solution of potassium ferrocyanide. It is difficult to say whether they grow these branches by the absorption of water—as chromatophores—or whether the processes are those of the connective tissue cells merely made visible by the presence of melanin. The cell bodies averaged 2 microns in length, and with their processes, they averaged 73 microns.

Sometimes granules of pigment may be seen peripherally located in the large round wandering cells. The pigment was found to be slightly soluble in hydrogen peroxide. Sections left in this solution for twenty-four hours showed fewer chromatophores, and those still present were light yellow instead of the dark brown characteristic of heavily pigmented

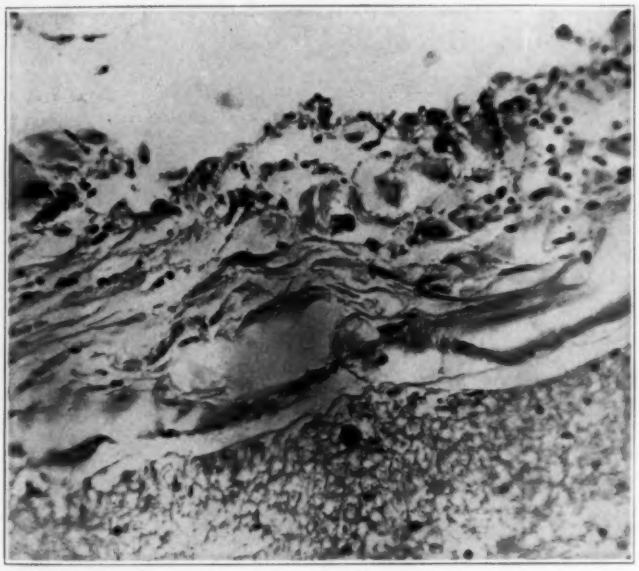

Fig. 6.—Pigment in the arachnoid of the cord.

specimens. After forty-eight hours most of the pigment cells had become completely invisible, and the ones that remained were pale yellow.

Pigment cells in the lower mammals differ from those in man in that the cells have more rounded bodies and fewer anastamosing branches. In many instances the pigment was dispersed in diffuse granules, no single cell type appearing as its bearer. In the monkey large ameboid-looking pigment cells were sometimes observed in the stria. These measured 5.8 microns. The granules were much coarser than those of the human being, but like the latter tended to aline themselves in parallel rows. Pigment cells found in the epidermis of the external auditory meatus in the monkey appeared to be shaped much more like those in the human cochlea.

In the rabbit huge globular cells, 10 microns in diameter, were present along the stria as bearers of pigment. Diffuse granules were also present. Branched pigment cells occurred at the bases of the maculae.

THE SIGNIFICANCE OF PIGMENT IN THE INNER EAR

The significance of melanophores in the labyrinth is not known. They may merely represent an embryonic remnant of a primitive pigmented vascular sheet (Acton) or revert to a time when the central

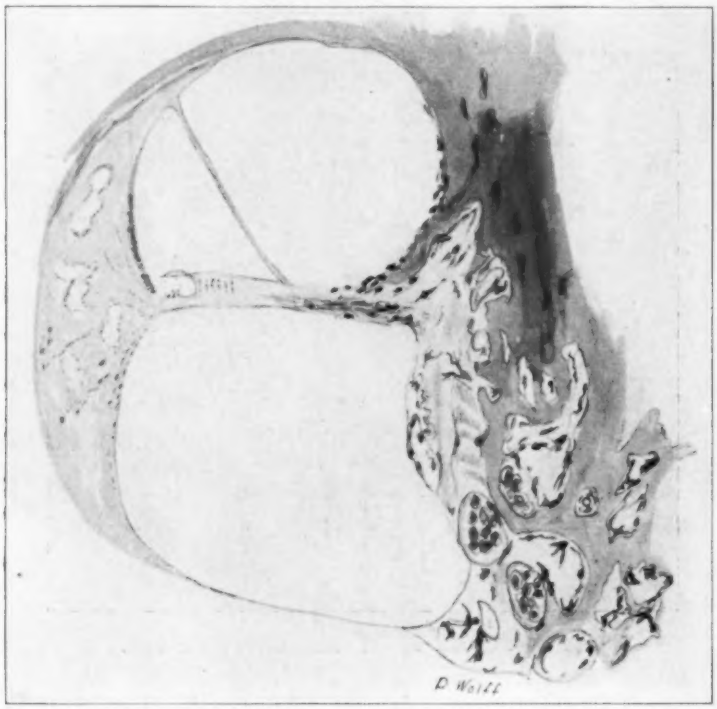

Fig. 7.—Distribution of pigment in one turn of the cochlea. (Water color.)

nervous system as a whole was encased in a pigmented sheath. Such a theory alone would correlate the occurrence in the brain, eye, ear, suprarenal gland and skin. No one can have been a student at Woods Hole without having observed and been fascinated by the varied chromatophores of the marine forms: the red pigment granules of the chorion of the sand dollar; the varicolored (red, yellow, blue, etc.) chromatophores of the developing squid, appearing and disappearing, and the melanophores of the fundulus, controlled, as Wyman described, by the nervous system. Do those of the squid represent some constantly reversible chemical action? They act like the play of an electric current.

VIEWS ON THE NATURE OF MELANIN

Arey, in speaking of the melanophores of the eye, said: "It is puzzling to determine whether the pigment processes are protruded and withdrawn like pseudopodia, or whether pigment granules flow into the processes."

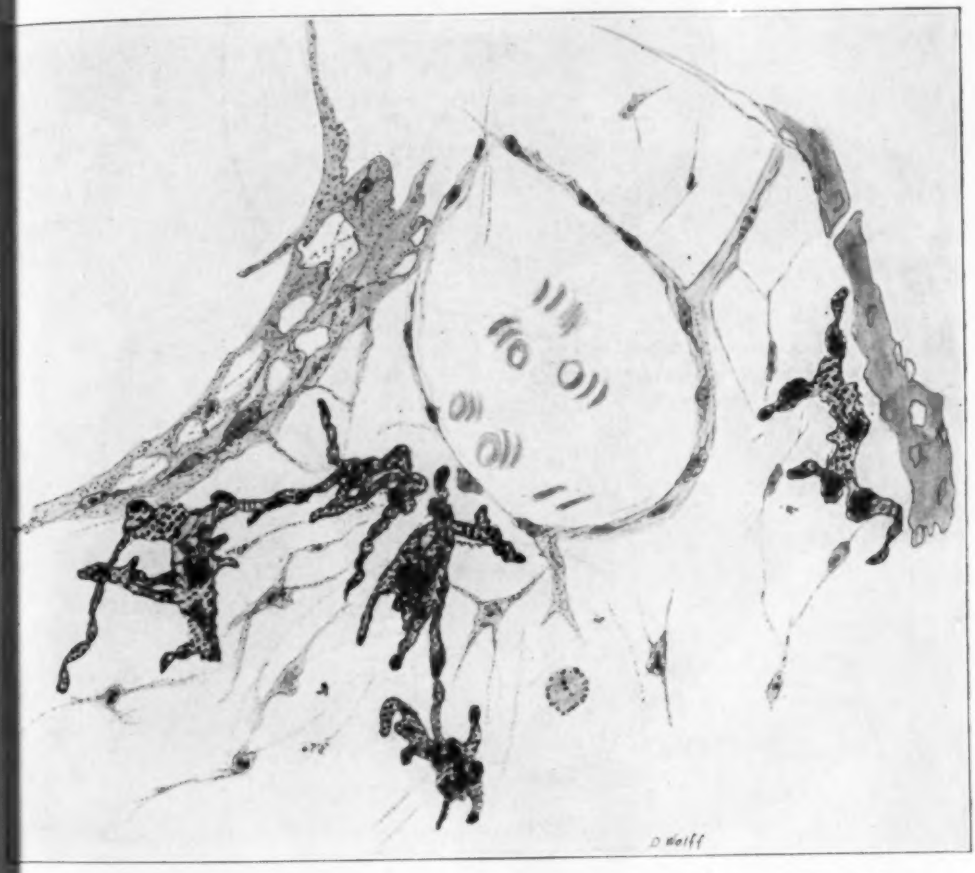

Fig. 8.—Pigment cells as seen in perivascular tissue of the modiolus. Oil immersion. (Water color.)

Bruno Bloch, professor of dermatology at Zurich, has extracted a substance—dioxyphenolalanin (dopa)—from the embryo of the broad bean. Frozen sections of fresh tissue treated with a 1:1,000 solution of dopa will exhibit melanin where melanogen, its forerunner, is present. This reaction, according to Spencer, does not occur in melanophores but only in melanoblasts, melanophores being the connective tissue bearers of pigment while melanoblasts are the manufacturers of melanin.

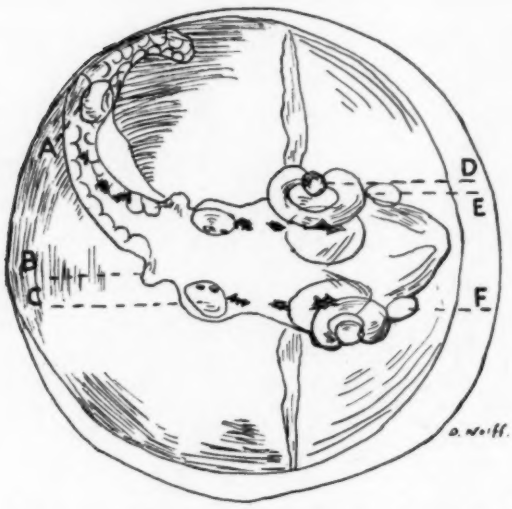

Fig. 9.—Fundulus heteroglitus (5 day fetus), showing yolk almost consumed; also alinement of pigment granules. *A* indicates Kupffer's vesicle; *B*, gill slit bud; *C*, otic capsule; *D*, lens; *E*, eye, and *F*, nasal capsule.

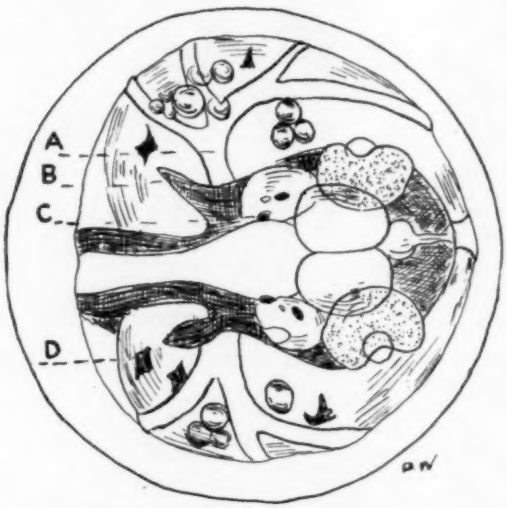

Fig. 10.—Fundulus majalis (7 day fetus), showing a decrease in the size of the yolk. *A* indicates the vitelline arteries; *B*, gill fringe; *C*, otolith, and *D*, pigment cell.

Melanin is believed by Bloch to be a protein-derived substance which is transformed by an enzyme oxydase into melanin. Gortner (1911) expressed the belief that there are two kinds of melanin. The melanoproteins are those soluble in weak acids and are not commonly found in pigment granules, though Gortner found them in auburn hair. The pigment granules that are not soluble in weak acids are oxidized from a different chromogen than melanoproteins.

The following paragraph is an abstract presenting the views of Pryde (1928) in regard to the chemical nature of melanin: The constitution of melanin remains undetermined. According to the Dumas

Fig. 11.—Developing squid with red and blue chromatophores.

methods it contains 8.65 per cent nitrogen. Tyrosinase is an oxidizing enzyme widely distributed in vegetable tissues. Its specific substrate is the amino-acid tyrosine which, in contact with the enzyme and in the presence of atmospheric oxygen, is oxidized ultimately to a black pigment melanin. When tyrosine acts on tyrosinase, a series of color changes occurs before the black insoluble melanin separates from the solution. First, at a p_H of from 6 to 8 a red pigment is formed, probably quinone. This is in turn decolorized and then oxidized to form melanin.

CONCLUSIONS

1. The membranous labyrinth should be included in the list of depots for branched melanophores along with the retina and choroid coats of the eye, the arachnoid of the brain and cord, the epidermis, etc.

2. In human embryonic life, pigment does not appear in the ear as soon as it appears in the eye.

3. Melanin was not found in the human fetal skin up to 6 months—not even in the Negro.

4. All Negroes, including stillborn infants, show pigment in the labyrinth. The age when it first appears is not yet known.

5. Among white persons more males than females show pigment.

6. In human beings the heaviest pigmentation is along the medial wall of the scala vestibuli and in the interstices of the cribriform modiolus.

7. In human beings the pigment is contained in large branching, anastomosing, connecting tissue-like cells.

8. In the animals examined, pigment occurs in the stria vascularis, if at all, and never in the interstices of the modiolus.

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News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Los Angeles on May 27, 1931, during the session of the Pacific Coast Society. Seventy-seven candidates were examined, of whom fifteen were conditioned or failed.

An examination was also held in Philadelphia on June 8, during the meeting of the American Medical Association. Ninety-eight candidates were examined, of whom eight were conditioned or failed.

The Board will hold an examination in Indianapolis on September 12, just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology, which will be held at French Lick, September 14 to 19. Prospective applicants for a certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, for proper application blanks.

H. P. Mosher, M.D., *President.*

W. P. Wherry, M.D., *Secretary-Treasurer.*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The American Academy of Ophthalmology and Otolaryngology will hold its Thirty-Sixth Annual Meeting at French Lick, Ind., Sept. 14 to 19, 1931.

Obituary

ROBERT CLYDE LYNCH, M.D.
1880-1931

Robert Clyde Lynch, one of the acknowledged leaders in the specialty of diseases of the nose, throat and ear, met a sudden and untimely death while driving from Berea, Ky., to Lexington with his old friend and colleague, Dr. J. A. Stucky of Lexington. In his death the South has lost one of the outstanding members of the medical profession, and otolaryngology one of its foremost workers and contributors. Dr. Lynch was what might be called a clinical investigator, while his operative technic and the clearness of his writings were a source of inspiration to his colleagues and the post-graduate students with whom he came in contact.

Dr. Lynch was born in Carson City, Nev., in 1880, and when 3 years of age he was taken by his parents to New Orleans, where his father was associated with the United States mint. He graduated in medicine at Tulane University in 1903, serving there as extern and intern in Charity Hospital, as well as student demonstrator in anatomy. In the latter position he no doubt gained much of the fundamental anatomic knowledge that was to be of such help to him in his later surgical work. Subsequent to his graduation, he entered general practice in Natchitoches, where he remained until 1905, accumulating thereby sufficient capital to pursue his studies in the specialty that he wished to follow. He then returned to New Orleans for post-graduate work. This was followed by further study in London, Paris, Vienna and Freiburg under Killian. Returning to New Orleans in 1907, he became associated with the Eye, Ear, Nose and Throat Hospital and in his private practice with Dr. Arthur W. De Roaldes, then the leading otolaryngologist of the South. On Dr. De Roaldes' death, he was made surgeon in charge of the nose, ear and throat department of the Eye, Ear, Nose and Throat Hospital and surgeon in chief of the same hospital in 1931. In addition, he was professor of otolaryngology in the Post-Graduate School of Medicine of Tulane University and consultant to the Touro Infirmary.

Dr. Lynch's contributions to the subject of otolaryngology were varied and continuous. His papers and writings always commanded the respect and attention of his colleagues, for they were founded on a keen observation as well as a vast and sound clinical experience. The living human body was his laboratory. Backed by a sound fundamental

training and inspired by a deep sympathy for his patients, his keen insight made him quick to understand the causes for his successes, as well as the reasons for his failures. He was the ideal clinician.

In addition to his positions on the editorial boards of the *Archives of Otolaryngology* and the *Annals of Otology, Rhinology and Laryngology*, he was a member of the American Board of Otolaryngology. He was also a member of the American Laryngological Association, the American Rhinological, Otological and Laryngological Society, the American

ROBERT CLYDE LYNCH, M.D.
1880-1931

Academy of Ophthalmology and Otology, the American College of Surgeons, the Southern Medical Association, the Louisiana State Medical Association, the Orleans Parish Medical Society, the New Orleans Ophthalmological and Otological Societies and of the American Medical Association. He belonged to practically all of the social clubs in New Orleans.

Among his outstanding achievements were his work on suspension laryngoscopy, fluoroscopic bronchoscopy and esophagoscopy, the Lynch

operation on the frontal sinus, his extensive experience on cancer of the larynx, with over 150 laryngectomies and his pioneer work on moving pictures of the vocal cords.

Medicine has lost an earnest worker, a keen observer, a tireless contributor. We who knew him best, in the various societies and in his clinic, and who have shared his generous hospitality in his home, have lost a lovable companion, a true and loyal friend, an inspiration in our work. His memory will be cherished by his students, his colleagues and his patients, in whom was his greatest interest.

Abstracts from Current Literature

Ear

MASTOIDITIS AND LATERAL SINUS THROMBOSIS IN AN INFANT. L. H. SEGAR and W. STOEFFLER, J. A. M. A. **96**:678 (Feb. 28) 1931.

The authors report a case of mastoiditis in an infant aged 8 weeks. The right tympanic membrane appeared normal in spite of the presence of mastoiditis. Lateral sinus thrombosis was the cause of death.

BONAR, Salt Lake City.

THE INTRA-AURAL ADMINISTRATION OF CERTAIN BACTERIA ASSOCIATED WITH MIDDLE EAR DISEASE IN ALBINO RATS. J. B. NELSON, J. Exper. Med. **52**:873, 1930.

The infective capacity of three bacteria commonly encountered during a study of natural disease of the middle ear in a rat colony has been determined by direct intra-aural injection into young rats. One week after the introduction of *B. actinoides*, 75 per cent of the rats showed a purulent exudate in the middle ear cavity into which the injection was made, and 65 per cent yielded pure cultures of the organism. With hemolytic and nonhemolytic streptococci, 75 per cent showed a serous or mucoid exudate, and 12 per cent yielded the organism in culture. With a diphtheroid, 18 per cent showed a gross reaction in the middle ear which was sterile in every case. The experimental observations are discussed in relation to the etiology of disease of the middle ear.

AUTHOR'S SUMMARY.

GENERAL OBSERVATIONS ON SEA-SICKNESS AND THE LABYRINTHINE THEORY. T. GWYNNE MAITLAND, Brit. M. J. **1**:171 (Jan. 31) 1931.

Various possible causes for sea-sickness are analyzed, and new data are presented to support the author's conclusion that the major cause of this disturbance is dependent on stimulation of the vertical canals. If the head is placed in such a position that the vertical canals are stimulated, symptoms can be produced experimentally by rotation. This corresponds to the movement of the ship, namely, the pitch, which is considered the important movement in the production of this phenomenon. However, three rotations are necessary. These are not sufficient to produce nystagmus, and therefore explain why nystagmus is not present in sea-sickness. Eight rotations, however, produce a period of rotatory nystagmus of six seconds, or vertical nystagmus of eight seconds, according to the position of the head, against fifteen seconds of horizontal nystagmus.

In regard to treatment, the author cautions against placing too much emphasis on any one form of therapeusis. The impression is gained that treatment should be considered as symptomatic and that there is no specific remedy.

FERGUSON, Niagara Falls, N. Y.

SYMPTOMLESS ACUTE MASTOIDITIS. N. ASHERSON, Lancet **1**:343 (Feb. 14) 1931.

The author cites four cases of symptomless acute mastoiditis, one of them in a child, aged 5 years. Sagging of the meatal wall was the only sign of a suppurative condition in the mastoid process or its extensions.

LANGMANN, New York.

THE DETECTION AND MEASUREMENT OF DEAFNESS IN SCHOOL CHILDREN BY MEANS OF A GRAMOPHONE AUDIOMETER. G. P. CROWDEN and A. H. GALE, M. Officer **44**:113 (Sept. 13) 1930.

The wide divergence in the recorded figures and in the records of the board of education concerning deafness among school children indicates a need for

standardization in the methods of examination. The usual forced whisper discloses gross defect, but in a room is too variable for early cases. The gramophone (4*A*) audiometer, with its standardized disks and magnetic pick-up, enables 40 children at a time to listen in with binaural telephones. The children transcribe the numbers that they hear dictated, and as a standard form is used to write on, it may be easily checked, and the loudness noted at which the children fail to hear well. The method excluded aid from unconscious lip reading. A nurse can obtain the records for the physician. In four Hornsey schools, 216 of 1,483 children tested failed at the first test, showing a loss of 9 or more sensation units. On retesting, 98 still showed this loss. In three of the schools, 50 of 69 examined showed: wax, 1 case; active disease needing immediate treatment, 13; remains of old disease, 21; and no abnormal appearance, 8.

The working of the audiometer is simple, and it draws attention to many cases, otherwise unnoticed, in which careful examination and treatment are required. Standardization for age and the psychic element of lack of apperception, however, are still needed for comparative purposes.

KUGELMASS, New York.

LESION OF BOTH AUDITORY NERVES, TOGETHER WITH SEVERAL OTHER CRANIAL NERVES ON THE LEFT SIDE. F. PARKES WEBER and M. SCHOLTZ, Proc. Roy. Soc. Med. **24**:107 (Dec.) 1930.

In a girl, aged 12 years, there was complete loss of function of both auditory nerves, including the vestibular portions, paresis of the facial muscles and soft palate on the left side, left lingual hemiatrophy with febrillary twitching, left recurrent laryngeal paralaysis and slight indistinctness in speech with slight dysphagia. There was slight nystagmus on looking to the left. She suffered frequently from headache over the right eyebrow. There was posterior polar cataract in right eye. The edges of the optic disks were somewhat indistinct. There were no other abnormal signs. The deafness was first noticed in December, 1929. In March, 1930, an alteration in speech was noticed. In August, 1930, twitching on the left side of the tongue was observed, and the child complained that the food stuck in her throat. There was a history of occasional slight epileptic seizure at six and a half years. Two suggestions were made as regards diagnosis: (1) bilateral tumors of the auditory nerve; however, on the left side there would have to be more than one tumor to account for the clinical symptoms; (2) syringobulbia.

WILLIAMSON, New Orleans.

THE LOCALIZATION OF THE VESTIBULAR REACTION OF THE EXTENDED ARMS AND OF THE POINTING TEST; THE DIAGNOSTIC VALUE OF THESE REACTIONS IN THE COURSE OF DISEASES OF THE CEREBELLAR FOSSA. G. S. ZIMMERMANN and A. S. TCHERNYCHEW, Rev. d'oto-neuro-opht. **8**:583 (Oct.) 1930.

In a case of tumor of the right side of the fourth ventricle, published in 1922, the right hand could not be made to past point to the left. This was thought to be due to hypofunctioning of the vestibular apparatus and not to involvement of Bárány's centers. The information gained from the case reported here will explain this phenomenon. In 1923, Barré repeated Bárány's chilling experiments. He believed that the thermic action was transmitted through the cerebellar cortex to the labyrinth by a vasomotor reflex. His conclusions were: 1. Past pointing is a vestibular, not a cerebellar, reaction. 2. Bárány's theory of cerebellar localization must be abandoned and his chilling experiments must be otherwise explained. In a series of cases of cerebellar lesions, Barré observed that the elements of the labyrinthine syndrome, which are present early and frequently in these cases, differ from those seen in pure labyrinthine disease. These anomalies are: lack of pastpointing with good nystagmus, deviation of the arms and nystagmus to one side and deviation of the trunk to the opposite side. In all the cases with anomalies of the vestibular syndrome Barré found other cerebellar signs: dysmetria

and adiadokokinesis. He concluded: 1. Disturbance of the cerebellar function may be manifested by disturbances of the normal vestibular syndrome. 2. When there is disharmony in the vestibular syndrome, a cerebellar factor must be looked for as the cause of it. The authors are in complete agreement with this view.

Bárány attributed a cerebellar localizing value to these anomalies, and almost all writers believe that an examination of the vestibular function gives information about the state of the cerebellum. This is not always true, because an anomalous vestibular reaction may be caused by a distant condition acting on the cerebellum.

De Stella expressed the belief that the functioning of the labyrinth is independent of the cerebellum. In his case of disease of the right cerebellopontile angle, after extirpation of the tumor, the hearing and the vestibular functions were reestablished and all disturbances of the cranial nerves disappeared, although the right cerebellar hemisphere was removed. The question arises, "What purpose is served by the pathway (cerebello-vestibular tract) that connects the cerebellum and the vestibular apparatus?" Stenver's theory was that the cerebellum constitutes a supplementary reflex pathway of the vestibular apparatus and that the development of this pathway is the result of long training. He based his theory on the facts cited by Edinger who traced the evolution of the cerebellum from the Myxines, which have no cerebellum, up through swimming fishes and birds. Destruction of these pathways causes the disappearance of the acquired functions: precision, harmony and perfection of movements. The case of Fischer and Pötzl, in which a large part of the right cerebellar hemisphere was removed, led them to believe that the cerebellum is not indispensable to the production of tonic vestibular reactions. However, the absence of its regulator function results in disharmony of vestibular reactions.

Sven Ingvar expressed the belief that there is not a distribution of function in the cerebellum according to the parts of the body, but according to different directions of movement and falling; the cerebellum receives and transforms the unconscious sensation of mass, and, by reflex action, it counteracts the impressions of the force of gravity and inertia in the body and thus preserves equilibrium.

The mechanism of past pointing is complex. When a person is asked to move the arm in a certain way, he must sense the direction, thus bringing the cerebral cortex into play. When different parts of the cerebrum are chilled, if the center of sensibility of direction is not touched, the subject will point correctly. The terminal cortical vestibular neuron probably ends at this point. When the excitation of this neuron passes a certain degree (rotary caloric tests), the normal sensation of direction is disturbed and the efferent impulse to the shoulder is not precise; at the same time the impulse from the stimulation is directed to the subcortical nuclei, especially those of the bulb and pons, and only partially to those of the cerebellum. These subcortical nuclei are directly connected with the anterior horns of the cord by the vestibulospinal pathway, and thus deviation of the extremities is produced. The central mechanism of past pointing comprises: (1) increase of tonus of the extremities in consequence of excitation of the subcortical centers from vestibular stimulation, and (2) modification of the cortical impulse because the movement is sensed. At times the cortical and at other times, the subcortical innervation is greater. The predominance of one or the other of these is revealed by the absence or the presence of deviation in the pointing test. This explains the different results obtained when the pointing test is used in normal persons.

In the case reported, the right cerebellar center for tonus of the movement of the arm downward was destroyed, the subcortical systems having been particularly injured, but the subcortical vestibular pathways in the pons and bulb were intact. Hence, the conclusion is evident that the reaction and the deviation of the right arm passed through the following reflex arc: vestibular nerve, nuclei of the bulb and pons, vestibulospinal pathway, and the anterior horns of the spinal marrow; not, as Bárány assumed, to the cerebellar cortex, central cerebellar nuclei, red nucleus and Monakow's bundle.

While adhering to the doctrine that vestibular deviation of the arm is not a cerebellar function, it is recognized that the cerebellum, through its anatomic

connections with the nuclei of the vestibular apparatus and with the cerebral cortex of the opposite side, may change the aspect of vestibular deviation. And, as the muscles of the trunk and the extremities are constantly subject to labyrinthine tonus, labyrinthine and retrolabyrinthine diseases that affect the bulb, the vestibulocerebellar pathways or the frontal or temporal cerebral regions may provoke spontaneous deviation of the arms; and, also, either directly or by acting from a distance, interference with any one of the elements of the reflex arc must cause deviation.

The anatomic hypotheses formulated by Ramón y Cajal and Kohnstamm, which supported Bárány's theory of localizations in the cerebellar cortex, are not confirmed by the recent researches of André-Thomas. According to the latter, only a few fibers of the root of the vestibular nerve go directly to the nuclei of the roof; a larger number originate in the nuclei of the vestibular nerve; the rare fibers of the bulb and pons are directed toward the cerebellar nuclei and enter the direct sensory pathway; the fibers of the vestibular nuclei go to the cerebellum and leave it as direct or crossed fibers. The uncinate tract (Russel) and the tectobulbar bundle (van Gehuchten) also unite the nuclei of the roof to the nuclei of the vestibular nerve. In spite of the intricacies of the two systems, it is possible to isolate vestibular from cerebellar symptoms. Labyrinthine spontaneous deviation (peripheral) is always accompanied by other labyrinthine symptoms, such as vertigo and horizontal nystagmus of small amplitude, but never by intracranial hypertension; it is habitually bilateral and directed toward the slow phase of nystagmus; at times, it is unilateral, but if so, there is a clear difference in the excitability of the two labyrinths. Spontaneous deviation of vestibular (retrolabyrinthine) origin is accompanied by an incomplete vestibular syndrome: labyrinthine vertigo is not always present, nystagmus is of wide amplitude and is more lively, and, in most cases, the deviation is directed toward the side of the lesion. When the lesion involves a considerable part of the cerebellum, especially the subcortical region, the vestibular syndrome is accompanied by typical cerebellar symptoms, such as ataxia, astasia, bradyteleocinesia or adiadokokinesis.

When the lesion is in the pathways of the middle or anterior cerebral fossae, vestibular symptoms are usually lacking. At times on stimulation (rotary, caloric) the rapid phase of nystagmus is absent, and spontaneous deviation of the arm is toward the side opposite the lesion. These observations are supported by the results of Groebell's experiments on pigeons.

On this basis the outward deviation of the left arm at the first examination in the case reported may be explained. According to Goldstein, cerebellar deviation is the result of the tendency to the movements of extension and abduction, which are automatic tendencies. Total removal of the cerebellum causes extension and abduction of the lower limbs; irritation causes adduction and flexion. This is also true of the cerebrum. Hence, the cerebrum and cerebellum inhibit automatic movements, and when this inhibition is removed by disease, spontaneous deviation is observed. Goldstein's ideas are confirmed by the authors' twenty-six cases. Of eleven cases with lesions in the cerebellopontile angle, spontaneous deviation was absent in six and present in five. Of nine cases with tumor of the cerebellum, deviation was present in five, doubtful in one and absent in three. In one case of abscess of the angle and in one of meningitis of the cerebellar fossa, spontaneous deviation occurred. In the twenty-six cases there were fifteen with spontaneous deviation (58 per cent). Of these, the deviation was toward the lesion in eight. Contrary to the opinion of many observers, this has some localizing value. According to Aubry, it is not often toward the side of the lesion. If the spontaneous and vestibular deviation is considered with reference to the seat of the lesion, Bárány's theory will be found in default in many. In the case reported, spontaneous deviation of the left arm outward, together with intracranial hypertension and slight cerebellar symptoms, points to a process acting on the cerebellar nuclei or on the pathways connecting them with the central vestibular system. The function of the regions damaged is to inhibit the reflex system; the right uncinate and the left entoconjunctival tracts are in relation with the left vestibular system. Hence, the important lesion on the right and the lesser one on the left resulted

in liberation of the left vestibular apparatus, with the consecutive appearance of automatic movements, i. e., deviation to the left. The disappearance of the spontaneous deviation at the last examination is explained by the compensatory action of the cerebrum. In the case of abscess in the angle, the deviation was upward. According to Bárány's theory, this would indicate a lesion in the posterior median pole of the cerebellum. The authors believe that it indicated disease of the cerebellar function in general and not a lesion of a definite region of the cortex.

In case 17, cold irrigation of the right ear did not reverse the spontaneous outward deviation of the left arm. This is explained as follows: "Disease of the uncinate tract and other pathways in the cerebellum damages inhibitory and reflex functions, causing spontaneous deviation of the index finger and arm in different directions." The stimuli directed to the labyrinth in this case were not powerful enough, or the vestibulospinal reflex was not in condition to overcome the deviation, which was due to abolishment of the inhibitory function of the cerebellum.

In case 7, the right arm did not deviate inward during cold irrigation of the right ear. This is explained by the fact that tumors of the angle cause necrobiosis in the region of Deiters' system (Tchernychew). The reflex arc (from the vestibular nuclei to the vestibulospinal tract) was damaged in this case, causing absence of the reaction.

The authors believe that they have simplified the clinical comprehension of these phenomena although they admit that, in the future, they may have to supplement or correct the theories advanced here. DENNIS, Colorado Springs, Colo.

HORIZONTAL NYSTAGMUS WITH CHANGING DIRECTION ACCORDING TO THE SUPPRESSION OF THE VISION OF EITHER EYE: TONIC AND KINETIC DISTURBANCES OF THE EYES FOLLOWING TRAUMATISM IN A PERSON WITH SYPHILIS. D. MANOLESCO, ST. DRAGONESCO and D. LAZARESCO, Rev. d'oto-neuro-opht. **8**:606 (Oct.) 1930.

A man, aged, 30, came to the clinic because of difficult vision. There was nothing significant in his past history except that his wife had had only one child, which was stillborn. Two years previous to admission, he received a blow on the occipital region and was in a comatose condition for three days. On recovering from the coma, he heard almost nothing in the right ear and his vision was reduced to the perception of light. He improved somewhat, but several days later he accidentally discovered that the right eye was blind. During three months he suffered with left hemicrania, which was worse in the frontal region.

Examination revealed a robust man with normal motility and coordination of the limbs. Hearing in the right ear was much reduced; in the Weber test the sound was not lateralized; the Rinné test gave negative results, and bone conduction was shortened on the right side. The pupils were normal. Visual acuity was reduced to one-half in the left eye and to perception of movements at 30 cm. in the right eye. The visual field in the left eye was contracted; the fundi were normal. Ocular motility was disturbed: With the right eye covered, movements of the left eye were normal; with the left eye covered, there was slight limitation upward and outward in the right eye. There were present: internal strabismus of the right eye; rapid and fine gyratory nystagmus in both eyes and a horizontal nystagmus to the left, which was intensified on looking to the left, and inability to turn the right eye completely to the right. When the left eye was covered, a horizontal nystagmus in the right eye to the right appeared, followed by marked internal deviation inward in the right eye; when the right eye was covered, there was a horizontal nystagmus to the left. There was no tonic deviation of the arms or the trunk.

A galvanic test of the ears, with the positive pole on the right, showed the following: with 2 ma. the head inclined to the right; with 4 ma. the arms deviated to the right, and with 8 ma. there was a horizontal and gyratory nystagmus to the left. If the right eye was then covered, the nystagmus was increased; if the left eye was covered, the right eye became immobile, but on increasing the

current to 12 ma., nystagmus to the left reappeared. With the positive pole on the left side, the same phenomena occurred, but they were reversed.

After irrigation of the right ear with 40 cc. of cold water, with the head backward at an angle of 60 degrees, there was horizontal nystagmus to the left, which changed to the right on covering the left eye; when 130 cc. of water had been used, the nystagmus to the right ceased, and after irrigation with 160 cc. of water, nystagmus to the left was reestablished. Deviation of the arms occurred following irrigation with 40 cc. of water. During the irrigation both eyes were converged, the right one markedly. Rotary nystagmus did not occur when the head was put forward 30 degrees. During irrigation of the left ear, pronounced internal strabismus of the left eye appeared at once; when 100 cc. of water had been used, the spontaneous nystagmus to the left ceased, to occur again feebly on covering the right eye; but on covering the left eye, a marked nystagmus to the right appeared; when 120 cc. of water had been used (both eyes being open), marked horizontal nystagmus to the right was seen; covering either eye changed this direction. When the head was tilted 30 degrees forward, rotatory nystagmus did not appear. Deviation of the arms was normal.

Rotation to the right caused intense horizontal nystagmus to the left, uninfluenced by covering either eye. Rotation to the left provoked a horizontal nystagmus to the right of lesser intensity, which could be neutralized by covering the right eye. Deviation of the arms was normal. Changing the position of the head and body suddenly on the same plane or turning the head to the right or the left caused a marked convergence of both eyes, followed in one-half minute by a nystagmus to the right.

Examination of the spinal fluid revealed: positive reactions to the Nonne-Apelt, Pandy and Bordet-Wassermann (3 plus) tests; 12.3 cells per cubic millimeter. The Bordet-Wassermann test of the blood was also positive. There were no neurologic signs of disturbances of the other cranial nerves.

Change in the direction of nystagmus and also inversion of nystagmus have been noted by numerous writers. The nystagmus to the right, appearing when the left eye was covered, was of central origin; it was intense, was not due to fixation (because it was present even when the good eye was covered) and was not a railroad nystagmus. It is difficult to understand the change of direction of the nystagmus in relation to covering one eye. The deficiency in the movements of the eyes upward and the spasmodic convergence both point to a lesion of the supranuclear pathways, especially of the posterior longitudinal bundle. Probably both the traumatism and syphilis are factors in the etiology.

DENNIS, Colorado Springs, Colo.

PARINAUD'S SYNDROME WITH LABYRINTHINE DISTURBANCES: THE PHYSIOPATHOLOGIC MECHANISM OF THIS SYNDROME. A. KREINDLER and A. SCHEIM, Rev. d'oto-neuro-opht. **8**:731 (Nov.) 1930.

This observation concerns a woman, aged 59, who, following an ictus, had a right hemiplegia with facial paralysis of the left side, paralysis of movements of the eyes upward, deviation of the right eye downward and outward, mydriasis in the right eye and spontaneous nystagmus. At the first examination, the urine contained no albumin or sugar, but later the sugar content was 3.6 per cent. Improvement soon began, and, as these clinical signs receded, cerebellar signs appeared: dysmetria, a tendency to fall backward and to the right and adiadokokinesis on the right.

Attention is called especially to the labyrinthine disturbances: spontaneous nystagmus, which disappeared after ten days, hyperexcitability to the galvanic current and hypo-excitability to the caloric test. They frequently occur in cases of associated paralysis of the eyes (Duverger and Barré; Léri and Bollak and Oljenik). Babinski, Vincent and Barré and Magnus regarded deviation of the eye downward and outward as a labyrinthine symptom. After extirpation of one labyrinth in a monkey, Bartels and Magnus observed a vertical deviation upward of the globe. Labyrinthine disturbances are not caused by the paralysis but by

reflex spasms of the muscles, originating in the labyrinth. Magnus and de Kleyn have shown that the distribution of tonus in the ocular muscles is regulated by tonic labyrinthine and neck reflexes, especially in animals with panoramic vision, but they also exist in man. The labyrinthine reflexes are exercised especially on the superior and inferior recti and the two oblique muscles; the neck reflexes, on the external and internal recti. These facts are useful in the interpretation of the physiopathologic mechanism of disturbances of associated movements of the eyes and the rôle that the labyrinth plays therein. In many, voluntary innervation of the ocular muscles is by way of the lower motor mechanisms, which are phylogenetically older and are in the nature of tonic labyrinthine and neck reflexes. In man, the tonic reflexes of the eyes are dissociated in the sense that functional association of the ocular muscles (vertical and lateral gaze) is made in the voluntary mechanism, independently of the position of the head in space. In cases of Parinaud's syndrome, the lesion affects the oculolabyrinthine association pathways. The mechanism of the tonic labyrinthine reflexes on the eyes is altered, and the voluntary innervation, coming from above, can no longer use this pathway to carry the eyes upward and downward. The alteration of the tonic reflexes in the neck on the eyes could, by analogy, be invoked in the pathogenic interpretation of Foville's syndrome.

DENNIS, Colorado Springs, Colo.

MIXED TUBERCULOUS AND MENINGOCOCCIC MENINGITIS. R. C. AGUIRRE and S. I. BETTINOTTI, Arch. argent. de pediat. **1**:611 (Dec.) 1930.

The authors report a case of mixed tuberculous and meningococcic meningitis in a child, 11 months of age, who was brought to the hospital because of a purulent discharge from both ears, coryza and cough, fever and anorexia, rigidity of the neck and a slight opisthotonus of two months' duration. Physical examination revealed a convergent strabismus, muscular twitching and convulsions, moist râles in both lungs, otitis media, splenomegalia and hepatomegalia. The tuberculin reaction was negative. Gram-negative diplococci with characteristics of meningococci were isolated from the pus of the ears and the cerebrospinal fluid; the latter also showed a definite lymphocytosis. The child died ten days later in spite of the treatment with antimeningococcic serum. Autopsy revealed the presence of a generalized miliary tuberculosis with caseo-ulcerative lesions in the apex of the lungs, meninges and mesenteric and tracheobronchial lymph nodes.

According to various statistical records, this type constitutes only 1.4 per cent of all forms of meningitis.

SCHLUTZ, Chicago.

THE SPEECH OF DEAF CHILDREN. A. LIEBMANN, Arch. f. Kinderh. **91**:288 (Oct. 21) 1930.

The author reviews his method of giving clarity to the faulty speech of deaf children by means of pictures. This should be begun by the time the child is 6 years of age.

SANFORD, Chicago.

ANSWER TO CRITICISM OF THE ELECTRICAL OTOSCOPE. HERBERT SCHEIN, Kinderärztl. Praxis **2**:39 (Jan.) 1931.

Schein defends the practical advantage of the electrical otoscope especially in the home.

HUENEKENS, Minneapolis.

MACULA UTRICULI ACCESSORIA IN HUMAN BEINGS AND OTHER CONGENITAL DEFECTS OF THE INTERNAL EAR. J. FISCHER, Monatschr. f. Ohrenh. **65**:517 (May) 1931.

Fischer begins his article by stating that congenital defects of the internal ear which do not impair its function are little known, and that in human beings they are rare. He then reviews the reports of several other authors as well as his own on such defects in animals and in human beings. All forms of congenital defects

of the internal ear which have been reported were found mainly in the medial, the bottleneck, portion of the utriculus and in the lateral wall of the sinus utricularis inferior. However, interesting, atypical epithelium formations on the macula utriculi of human beings have not been described as yet. The author had the opportunity to observe a large defect which involved the macula utriculi and the adjoining wall of the utriculus. The specimen was from a man, aged 28, who had died of pneumonia. The clinical history reveals that the patient had never been subject to ear disease. In order to explain the peculiarities of the case the author describes the results of the histologic examinations. He studied 160 sections. Following a classification of the results of these histologic examinations he evaluates the peculiar observations. The formation, in the bifurcation of the bony frame, of the tympanic membrane with its bony apophysis directed toward the chorda was atavistic. It occurs in many mammals. The unusually large ridges in the hypotympanum are known, but in the degree in which they appeared here, they may be considered as rare. The bulbous thickening on the tendon of the tensor tympani was likewise an atavistic formation. The membrane that occluded the approach to the secondary tympanic membrane was a rest of the embryonal mucous tissue in the tympanic cavity. The bone fissures were larger and more numerous than is usually the case. The bony cochlea was delicate. The lamina spiralis ossea secundaria was unusually wide and thin; in one place it appeared dehiscent. There was also a dehiscence in the facialis canal, and the fasciculation of the facialis was likewise abnormal. The endolymphatic protuberances on the semicircular canals were unusually numerous. The cyst formations on the membranous semicircular canals as well as the spherical inclusions in the epithelium of the cristae were likewise variations. In the pars superior as well as in the pars inferior there were atypical places in the epithelium characterized by a circumscribed absence of the hair cells, as the result of which the supporting cells were closer together. However, the most noteworthy defect involved the macula utriculi. The malformation consisted of two folds. They were so arranged that from the large utriculus cavity a smaller cavity was cut off by a circular wall. Considering the structure of the fold, it belonged to the macula utriculi and for this reason the author designates it as the macula utriculi accessoria. The neuro-epithelium of the macula utriculi accessoria merged into the epithelium of the macula utriculi and extended a little further into those portions of the fold which emerge from a small elevation of the utriculus wall where there are no terminations of nerves. Other malformations in the utriculus were: nodules on the medial wall, consisting of epithelium and of perilymphatic connective tissue cells, ayptical sites in the epithelium and cystic cavities filled with supporting cells and with exudate. The perilymphatic tissue of the other utriculus wall appeared more strongly developed. In the sacculus there was a ridgelike endolymphatic elevation. The macula sacculi was unusually large. The ductus and the sacculus endolyphaticus showed many variations. The circumscribed atrophy in a portion of the cochlear canal, the abnormalities in the blood vessels of the stria vascularis, the atrophy at the base of the stria and the absence of the vas prominens in the upper portion of the cochlea were likewise congenital anomalies. In conclusion the author states that there is no other report in the literature of a case with such marked congenital defects of the internal ear, which apparently did not impair the function. He considers the macula utriculi accessoria as an atavistic formation. So far as the possibility of a physiologic function is concerned, he considers it comparable to the lagena, even if topographically it has nothing in common with the macula lagenae.

EDITOR'S ABSTRACT.

THE BACTERIOLOGY OF OTITIS MEDIA FOLLOWING SCARLET FEVER AND DIPHTHERIA. S. KOPAČ, Časop. lék. česk. **67**:1777 (Dec. 28) 1928.

Of 198 cases of scarlet fever, 8 (4 per cent) were complicated by infection of the middle ear. Of 139 cases of scarlet fever and diphtheria combined, 10 (7.2 per cent) showed otitis media. Of 450 patients with diphtheria, 4 (0.8 per

cent) had a complicating otitis media. Virulent diphtheria organisms were found in 45 per cent of these, and were distributed as follows: in 50 per cent of the cases of scarlet fever, in 25 per cent of the mixed cases and in only 25 per cent of the cases of diphtheria. The question of a diphtheria carrier in otitis media becomes important because none of the cases with virulent diphtheria showed symptoms differing from those in ordinary otitis media. No membrane formation was found. In such cases a virulent discharge may be present for a period of years.

STULIK, Chicago.

Pharynx

THE RELATION OF TONSILS AND ADENOIDS TO INFECTIONS IN CHILDREN. ALBERT D. KAISER, Am. J. Dis. Child. **41**:568 (March) 1931.

After studying almost 5,000 children before and after tonsillectomy, Kaiser observed that chorea developed more frequently in children who had undergone this operation than in those who still retained tonsils and adenoids. The incidence of chorea among tonsillectomized children was 1 per cent; among unoperated children, 0.6 per cent. However, chorea in a tonsillectomized child is less apt to be followed by a cardiac complication than in a patient who has not undergone the operation. Tonsillectomy favorably influenced the incidence of colds in the head, sore throats, otitis, lymphadenitis and acute rheumatism. Bronchitis, pneumonia and sinusitis were more common in children whose tonsils and adenoids had been removed. From Kaiser's study it would appear that chorea is not an indication for the removal of healthy tonsils.

DAVIDSON, Newark, N. J.

HUMAN CARRIERS OF STREPTOCOCCUS EPIDEMICUS. I. PILOT, B. HALLMAN and D. J. DAVIS, J. Infect. Dis. **47**:503, 1930.

Streptococcus epidemics is found in the throats of persons during interepidemic periods. The chief habitat appears to be the crypts of the faucial tonsils. Three carriers of *S. epidemicus* had enlarged tonsils, cervical adenitis and arthritic pains. The recognition of these carriers is important because of their possible rôle in the transmission of the disease to others and particularly in the accidental infection of the udder of the cow, with subsequent explosive epidemic sore throat spread through the milk.

AUTHORS' SUMMARY.

THE RELATIONSHIP OF THROAT INFECTION TO ACUTE RHEUMATISM IN CHILDHOOD. B. SCHLESINGER, Arch. Dis. Childhood **5**:41 (Dec.) 1930.

This investigation is based on four years' clinical study in a hospital for children with rheumatic disease of the heart. Infection of the throat occurred in sixty-two cases, and twenty-two of these patients suffered a relapse of rheumatism. Bacteriologic study does not reveal any specific type of streptococcus responsible for the infection of the throat or the acute rheumatism.

The interval of time, from ten to twenty-one days, between the onset of tonsillitis and the rheumatic relapse is almost constant; the same interval is seen in scarlatinal rheumatism, and the whole problem is best explained on an allergic basis, comparable to serum disease. This by no means excludes a bacterial origin for rheumatic disease, since here a bacterial antigen is an essential precursor of the allergic state. Far from supplanting the theory of streptococcal origin, an allergic theory helps to strengthen the view that this group of micro-organisms is the exciting cause of the disease.

There is as yet no means of estimating a patient's power of resistance against further attacks of rheumatism. Dick tests only show that rheumatic children behave like the rest of the normal population and, further, that rheumatic children occur irrespective of the reaction of the patient to this particular toxin. Cutaneous tests with filtrates of other streptococci are also inconclusive.

Prophylaxis is naturally the ultimate aim, and tonsillectomy would seem to be the most obvious course. Unfortunately the operation, apart from giving some protection against epidemic sore throat, has repeatedly been shown to exert little influence in other ways on the course of rheumatism. Some evidence has been produced showing that the rheumatic patient with established cardiac disease is often better served by septic tonsils than by a healthy throat. The operation should therefore be reserved for those children whose general health is being adversely affected by this chronic focus of infection.

Prolonged convalescence in a special country hospital has been one of the recent advances in the treatment of children with acute rheumatism and early cardiac disease. Epidemics of rheumatism in homes of this nature have been described, in which a preceding infection was invariably disclosed. It appears that this rather than the rheumatism itself was the infectious element.

The problem is centered on the pharynx; it seems that if outbreaks of throat infection could be avoided, rheumatic relapses would practically never occur. To banish these infections during the winter months seems impossible at present; the most that can be done is to limit the infection by means of small wards and strict isolation.

Acetylsalicylic acid given for several weeks after the onset of tonsillitis has proved to be a valuable method of preventing the recurrence of serious rheumatism.

KELLY, Milwaukee.

THROMBOPHLEBITIS OF THE CAVERNOUS AND INTERCAVERNOUS SINUSES: BILATERAL ORBITAL THROMBOPHLEBITIS CONSECUTIVE TO PERITONSILLAR ABSCESS. EEMAN, Rev. d'oto-neuro-opht. **8**:744 (Nov.) 1930.

This case report emphasizes the fact that peritonsillar abscess is a dangerous condition. The patient showed a typical picture of cavernous sinus thrombosis and died four days after admission. The venous connections are described as follows: "The orbital veins communicate in front with the facial veins, in back with the cavernous sinus and the cerebral veins, and below with the pterygoid plexus." Infections of the face, mouth or pharynx, especially of the nose and eyelids, are the common cause of thrombosis of the cavernous sinus. The prognosis, which is grave, and the treatment, usually ineffective, are discussed.

DENNIS, Colorado Springs, Colo.

ACUTE MYCOTIC TONSILLITIS: OBSERVATIONS ON A CASE OF ACUTE TONSILLITIS RESULTING FROM MONILIA METATROPICALIS IN A BOY. A. MUGGIA, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 265.

A child, aged 15 months, contracted a sudden illness with fever. Examination showed a sore throat. The follicles of the tonsils were filled with a milk-white exudate. Microscopic examination of the exudate showed only *Monilia*. The child had a high fever (from 39 to 40 C. [102.2 to 104 F.]) for five days; within three days the temperature had become normal. The exudate was scraped easily from the tonsils, but reformed rapidly; by the fourth day of the disease it covered almost the whole mouth. In a short while, it completely disappeared. The author made exhaustive microscopic, cultural and biologic studies of the organism, which he classified as *Monilia metatropicalis*. This has been described by Castellani (*J. Trop. Med.*, 1923).

HIGGINS, Boston.

THE TECHNIC OF TAMPONADE FOLLOWING TONSILLECTOMY. F. NOLTENIUS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:50 (June 6) 1931.

Noltenius states that for the last eight years he has employed the so-called "tampon suture" for primary arrest of hemorrhage. Originally he had used it for secondary arrest of hemorrhage, but because of the good results he obtained with

it and also because he was forced to perform most tonsillectomies in ambulatory patients, he followed the tonsillectomy immediately by the tampon suture. As the advantages of this treatment he gives the following: 1. Since he introduced this method he has never had a postoperative hemorrhage. 2. The subjective postoperative symptoms are lessened. With few exceptions the spontaneous pain has generally disappeared on the third day. The author thinks that this is due to the fact that the iodoform gauze tampon prevents inflammation of the wound and thus also pain. 3. For the same reason the course of healing is more rapid and less complicated. The tampon generally remains for from forty to forty-eight hours. A hemorrhage during the second week following the intervention cannot, of course, be prevented by the tampon suture, but it can usually be prevented by a careful surgical technic. The author gives a detailed description of the technic of the tampon suture and asserts that it is simple. He also describes the removal of the tampon and states that in 120 cases he did not note injurious effects from the primary suture.

EDITOR'S ABSTRACT.

THE TONSILS IN CORRELATION WITH THE THYROID. JASTREBOWA, Monatschr. f. Ohrenh. **65**:576 (May) 1931.

In order to determine the influence of tonsillectomy on the thyroid, Jastrebowa performed experiments on young pigs. On the basis of her observations she reaches the following conclusions: 1. The growth of young pigs which have undergone tonsillectomy is retarded in comparison with that of control animals in which the tonsils have not been extirpated. 2. Tonsillectomy causes marked changes in the tissues of the thyroid. 3. Removal of the tonsils causes changes not only in the tonsillectomized animals themselves but apparently also in their offspring, in the form of congenital weakness and changes in the thyroid. 4. Observations on two animals indicated that resistance against infection is weakened even by partial tonsillectomy. The author concludes that the results of these experiments justify the negative attitude of their clinic toward tonsillectomy. They consider that it is not an indifferent intervention and that it is therefore inadvisable except when special indications exist, as, for instance, in case of neoplasms.

EDITOR'S ABSTRACT.

DEVELOPMENT OF ENDOCRANIAL COMPLICATIONS AFTER TONSILLITIS AND PERITONSILLITIS. E. WESSELY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:167 (May 26) 1931.

Wessely first directs attention to the reports of other authors as well as to his own previous investigations and observations on the development of endocranial complications after tonsillar processes. He mentions particularly the tests on corpses which he conducted several years before. By making injections of india ink into the peritonsillar tissues of corpses shortly before death, he had noted the transmission of the india ink through the spatium parapharyngeum up to the foramen ovale. This route seemed to be best suited anatomically on account of the looseness of the connective tissue, and it also explained all symptoms on the part of the musculature and the nerves. In the main part of the article the author gives several new case reports and describes the results of his studies of these cases. In order to make a detailed study of the route of infection in one case he removed at autopsy a frontal block consisting of the left tonsillar region, the corresponding portions of the mandibula and of the base of the skull, prepared it for histologic examination and examined 280 sections. After describing the results of his studies he states that the process was a phlegmon with perforation into the venous system. The inflammatory spreading took place in the anterior portion of the pharyngeal space upward, and attained its strongest development at the base of the skull. Here the pterygoid muscles and the loose connective tissue of the spatium parapharyngeum contained abscesses and in portions they were destroyed. From the anterior edge of the internal pterygoid muscle the process was transmitted by suppuration along

the branches of the trigeminus and also along the middle meningeal artery, through the foramen spinosum, into the middle cranial fossa. This is the route which seems predestined by the loose connective tissue and by the direct transmission along the nervous and arterial channels. This case is typical and many other cases are similar to it. However, another case described by the author represents apparently an entirely different type. In this case there existed a peritonsillar abscess, which did not perforate into the mouth but eroded the pharyngeal wall. During a fever-free interval a second abscess formed, which spread in the direction of the least resistance, namely, cranialward. The inflammatory irritation of the nerves caused toothaches and later trigeminus neuralgia. On the other hand, in the plexus pterygoidens veins became thrombosed, eroded and infected, and gradually a retrograde thrombosis of the cavernous sinus developed with typical ocular symptoms. However, the fully developed pathologico-anatomic aspects in this instance were similar to those observed in the other case, only the course of the development was much slower and the temperatures were lower. EDITOR'S ABSTRACT.

I. DISTURBANCES IN CARBOHYDRATE METABOLISM IN THE COURSE OF TONSILLAR DISEASES. L. KOSTYÁL, Ztschr. f. Kinderh. **50**:231, 1930.

Starting with the fact that in diphtheria there is a lesion in the liver that leads to a disturbance of carbohydrate metabolism, Kostyál studied fifty children. In all the cases of faucial diphtheria, the blood sugar was increased; in two thirds of the cases, the valve immediately increased to over 100 mg. per hundred cubic centimeters. The occurrence, duration and extent of the hyperglycemia were in no way related to the severity of the local disease. With lymphadenitis and tonsillitis there was in some cases a diminution of the blood sugar, even a hypoglycemia, which either remained unaltered or changed to normal values or hyperglycemia. In croupous laryngitis plus diphtheria, the blood sugar value is like that in faucial diphtheria; in croup plus tonsillitis, the content is like that in tonsillitis, or is between the values for the two diseases. Except in complications, the disturbed function of the liver and endocrine apparatus is held to be responsible for the disturbed carbohydrate metabolism. In scarlet fever hyperglycemia occurs, proportional to the degree and duration of the disease; with the decrease of fever and blanching of the exanthem, the blood sugar level falls; this may be due either to a sympathicotonia or to a general endogenous disturbance of equilibrium. In tonsillitis the blood sugar decreases; there may be even a hypoglycemia. After the first day of the acute stage the sugar content may be normal.

EMERSON, Boston.

II. DISTURBANCES IN CARBOHYDRATE METABOLISM IN THE COURSE OF TONSILLAR DISEASES. L. KOSTYÁL, Ztschr. f. Kinderh. **50**:240, 1930.

In the acute stage of scarlet fever and diphtheria there is a sympathicotonia; in tonsillitis, a vagotonia. The state of the vegetative nervous sytsem is independent of fever, the duration of the disease and the severity of the local changes. In the convalescent stage of scarlet fever the sympathicotonia changes to vagotonia; in diphtheria, it remains as it is, and in tonsillitis the vagotonia changes to sympathicotonia. In these diseases there is doubtless a disturbance in the vegetative nervous system, differing qualitatively, although in all three it can be referred to an endogenous disturbance of equilibrium.

EMERSON, Boston.

III. DISTURBANCES IN CARBOHYDRATE METABOLISM IN THE COURSE OF TONSILLAR DISEASES. L. KOSTYÁL and A. TAKÁCS, Ztschr. f. Kinderh. **50**:243, 1930.

Liver function was tested by the use of galactose and levulose. Study fortified the assumption that in tonsillar diseases there occurs a simultaneous injury to the parenchyma of the liver and its function. The disturbance in carbohydrate metab-

olism is least in scarlet fever, greatest in tonsillitis and between the two in diphtheria. Doubtless the glycogen metabolism in the liver is injured. The question remains open whether the liver is injured directly, or indirectly through the endocrine glands. It is of interest that except in tonsillitis, the hyperglycemia was the same after the administration of galactose and levulose. Traces of glycosuria were noted after the use of levulose in only five cases. Apparently the kidneys have greater permeability for levulose than for galactose in these diseases.

EMERSON, Boston.

Larynx

ULCERATIVE LARYNGITIS DUE TO CORYNEBACTERIUM ULCERANS. JESSE G. M. BULLOWA and MENDEL JACOBI, Am. J. Dis. Child. **41**:120, 1931.

A case of fatal ulcerative laryngitis due to an atypical diphtheria-like organism and producing only a slight fibrinous reaction is reported. A culture of the organism from a laryngeal ulcer is identified as the *Corynebacterium ulcerans* of Gilbert and Stewart, previously isolated only from cultures in nonfatal infections of the upper respiratory tract. Both organisms produce extensive necrosis and ulceration without the formation of typical diphtheritic pseudomembrane. The immunologic study of cases of diphtheria not responding to antitoxin is suggested.

AUTHORS' SUMMARY.

TREATMENT OF TUBERCULOUS ULCERATIONS OF THE LARYNX BY MEANS OF ACRIFLAVINE HYDROCHLORIDE. D. DÖRNER, Monatschr. f. Ohrenh. **65**:569 (May) 1931.

After discussing the history and the therapeutic action of acriflavine hydrochloride Dörner points out that the favorable results obtained with it in tonsillitis and in surgical, suppurative processes induced him to try it in tuberculosis of the larynx. He gives eight case reports, which indicate that acriflavine hydrochloride has a favorable influence on ulcerative laryngeal tuberculosis. The action was not the same in all instances. In some cases the results were excellent and permanent, whereas in others there was only a temporary improvement. In order to explain this, attention is directed to the fact that laryngeal tuberculosis has various forms of manifestation, the four main types being the lupus form, the tumor form, the disintegrating proliferative form and the torpid ulcerative form, and it is understandable that the therapeutic action of the acriflavine hydrochloride varies in these different forms. In regard to the application of the acriflavine hydrochloride, it is stated that at first the lesions were powdered by means of an insufflator, but later it was found that this way of application is not always successful. In cases in which the ulcerations are deep, covered with mucus and coated, the acriflavine powder has to be rubbed in so that it reaches every recess. This is sometimes difficult, but if it is done right, one application weekly is generally sufficient. The author thinks that the treatment with acriflavine hydrochloride is superior to all other therapeutic procedures. It has the advantage that it effects freedom from pain not by destroying the nerve or the tissues, but rather by destroying the pathogenic micro-organisms and by clearing the infectious foci and the ulcerating processes. Even in cases in which healing does not set in, treatment with acriflavine hydrochloride protects the organism against the danger of mixed infection. Harmful secondary effects of the treatment were never noted. About the action of acriflavine on closed foci a definite statement cannot be made as yet, but observations so far indicate a favorable influence. In order to determine whether the acriflavine influences only the bacteria of the mixed infection and the suppurations and painful inflammations of the tuberculous processes or whether the tubercle bacilli are also influenced, experiments on animals have been begun. On the basis of his experiences the author recommends the use of acriflavine hydrochloride for the treatment of laryngeal tuberculosis. EDITOR'S ABSTRACT.

STENOSING MALFORMATION OF THE LARYNX: THREE CASES. K. SCHRÖDER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:182 (May 26) 1931.

The first case described by Schröder concerns a nursling. When 11 weeks old the infant was brought to the clinic with stridor, dyspnea and cyanosis. It was stated that stridor and dyspnea had existed since birth and that the infant had never cried and could not swallow easily. To overcome the dyspnea a tracheotomy was done, but the child died on the following day as the result of pneumonia. Autopsy revealed bilateral bronchopneumonia, an insufficient development of the lungs, probably as the result of the inhibited respiration, and, besides the stenosis of the larynx, a stenosis of the right ureter. Careful examination of the larynx revealed that the rima glottidis, with the exception of a small opening at the lateral wall, was entirely closed. Histologic examination revealed that the tissues which formed the closure consisted of the fused cricoid cartilages, muscles, mucous glands and fat tissues. A second case of malformation of the larynx, which differed from the first one only in degree, concerned a man, aged 23. The patient stated that he had been hoarse since childhood and that he had difficulty in breathing. Examination revealed that the rima glottidis was two-thirds closed, apparently by a fusion of the vocal cords. A laryngofissure was done and the coalescence was divided in the direction of the rima glottidis. The author describes other details of the operation and states that at first the patient's voice was hoarse, but the breathing was much freer; a year after the operation, the patient was able to take part in strenuous athletic activities and his voice was normal. After discussing the probable embryonal causes of these malformations of the larynx the author gives a third case report concerning a woman, aged 53. She had severe dyspnea and stated that she had never had a clear voice. She had had severe attacks of dyspnea twice before, once during childhood and once at the age of 40. A renewed attack now induced her to consult a specialist, who detected a cyst which closed the opening of the larynx almost completely and which was attached to the right aryepiglottic fold. Repeated incisions yielded first a mucous fluid and later a suppurative fluid. However, the opening always closed again and the cyst refilled. The patient was therefore sent to the clinic for the purpose of a more radical operation. The author describes the operation and states that the patient died several days later as a result of pneumonia. He discusses the results of the histologic examination of the larynx, and in the conclusion he points out that a different surgical method might have prevented postoperative pneumonia and the death of the patient. He thinks that for the removal of a cyst, with a location like that in the reported case, von Hippel's operation for an infected intralaryngeal sac would be advisable.

EDITOR'S ABSTRACT.

Nose

THE RELATION OF THE PARANASAL SINUSES TO OCULAR DISORDERS, ESPECIALLY TO RETROBULBAR NEURITIS. S. R. GIFFORD, Arch. Ophth. **5**:276 (Feb.) 1931.

This article is an ophthalmologic review of the literature. In the first part of the paper, the cases presented by various authors are analyzed from one standpoint alone: Were the ocular disturbances caused by disease of the paranasal sinuses or was the etiologic factor an entirely different causative agent? The second part of the review is a discussion of the pathology of the type of retrobulbar neuritis that is caused by disease of the sinuses: the acuteness or the severity of the morbid process; the path of the infection (hematogenous or by contiguity of the tissues); the microscopic changes; the complications that arise, as pneumonia, thrombosis of the cavernous sinus, etc., and the importance of a histologic examination of the bone in disease of the nasal accessory sinuses.

The third part of the review deals with the indications for operation. When one considers the surgical treatment for retrobulbar neuritis one must of necessity eliminate all probable causes other than disease of the sinuses, especially multiple sclerosis, tumor of the brain and amblyopia due to tobacco or alcohol. Only

cases can be included in which definite purulent sinusitis is present. The time for surgical intervention, the preoperative treatment and the operative results are discussed. The author calls attention to the importance of field studies in determining the advisability of and the time for operation.

In the summary of the article, the author again emphasizes the facts that 50 per cent of cases of retrobulbar neuritis are due to multiple sclerosis, that purulent sinusitis is responsible for about 3.5 per cent of cases, and that in from 15 to 20 per cent of the cases in which no cause can be found opinions still differ as to how many are due to latent or hyperplastic sinusitis. Pathologic evidence seems to show that retrobulbar neuritis may be caused by direct extension. Before operation on the sinuses, every effort should be made to eliminate all other probable causes for the ocular disorder. It is only in this way that accurate statistics can be obtained. The author advises, furthermore, that all tissue removed from the nose be examined histologically. Through a knowledge of the pathology of the morbid process in such cases a better understanding of the whole problem will be obtained. He believes also that there are a number of cases in which exploratory operations on the sinuses are indicated.

SPAETH, Philadelphia.

HEREDITARY EPISTAXIS; WITH AND WITHOUT HEREDITARY (FAMILIAL) MULTIPLE HEMORRHAGIC TELANGIECTASIA (OSLER'S DISEASE). HYMAN I. GOLDSTEIN, Internat. Clin. **3**:148 (Sept.); 253 (Dec.) 1930.

Essential features of hereditary epistaxis are: (1) the occurrence of nosebleed in childhood, often recurring during the life of the patient, sometimes associated with bleeding from other mucous surfaces; (2) the development of various forms of telangiectases; (3) the occurrence of the disease in several members of the same family in several generations. The number of blood platelets, bleeding time and clotting time are normal. Men and women are affected, and both sexes may transmit the condition. Detailed histories of three afflicted families are given.

MILLER, South Bend, Ind.

Miscellaneous

FACIAL NEURALGIA TREATED BY RETROGASSERIAN NEUROTOMY. AMZA JIANU, D. PAULIAN and D. TURNESCO, Rev. d'oto-neuro-opht. **8**:614 (Oct.) 1930.

A married woman, aged 56, entered the hospital with facial neuralgia on the right side. The past history was not significant. The pain, which had been present for six years, followed dental caries. At first it was localized in the right upper gum and was amenable to treatment with anodynes, but later it extended to the entire right half of the face. In spite of a variety of treatments, including two injections of alcohol, relief was only temporary. A resection of the sensory root of the trigeminus was done under local anesthesia, following a tarsorrhaphy. On exposure of the ganglion, a sharp hemorrhage occurred. The sensory root was severed, but the motor root was not identified. On account of the bleeding, a strip of gauze was packed in the depth of the wound and was led out through the lower angle of the incision, which was closed with catgut. The operation lasted three hours. The postoperative course was uneventful except for a rise of temperature to 39 C. (102.2 F.) on the second day and again in the period between the tenth and the sixteenth days. The pain was immediately relieved.

An examination of the patient two months later revealed a slight facial asymmetry but no paralysis; sensation was abolished in the domain supplied by the fifth nerve on the right; the corneal reflex was absent and the cornea was intact. The right masseter muscle was not paralyzed.

Injections of alcohol have a practical importance in the diagnosis of facial neuralgia and in the prognosis of pregasserian neurotomy. When the injections do not cause even a temporary amelioration of the pain, the condition is not essentially one of facial neuralgia, and neurotomy will be unsuccessful.

Of the three surgical methods of treatment, section of the branches, removal of the ganglion and section of the sensory root, the last is preferable. It can be made at two points: the neighborhood of the bulb, or just before its termination in the ganglion. Lesions of the masseter nerves and trophic disturbances of the eye from traumatism of the ganglion are dangers of the operation. Dividing the root near the bulb, in accordance with Dandy's proposal, has the advantages of greater visibility and the possibility of a partial division of the root. According to Dandy, division of three fourths of the root is sufficient to relieve the pain and the disagreeable sensation in the areas supplied by the nerves, and trophic troubles do not occur. On the other hand, this method of operation is much graver than the other methods mentioned, and there is danger of injuring the eighth nerve. The importance of severing the masseter nerve is not great, because, owing to the anesthesia in the mucosa, these patients will chew on the healthy side. Keratitis can be partially avoided by a tarsorrhaphy preliminary to the operation. Relapses are possible after partial resection and even after total division, but they are exceptional.

DENNIS, Colorado Springs, Colo.

A CONTRIBUTION TO THE STUDY OF HYSTERICAL APHASIA. GIOVANNI DI LALLU, Ann. di nevrolog. **44**:81 (May-June) 1930.

Hysterical disturbances in speech may be as numerous as those caused by organic lesions. The most common forms include simple aphonia, mutism, anarthria and dysarthria. True aphasic disturbances in hysteria are rare. According to some observers, all types of organic aphasia may be observed in hysteria. Cases of word blindness, word deafness, "aphemia" and agraphia have been reported.

The author describes a case of motor aphasia in a woman, aged 28. In addition to the aphasia, she presented typical hysterical right hemiplegia, right hemianesthesia and right homonymous hemianopia. All of these symptoms, including the aphasia, disappeared after the application of an electric current. The difficulty in speech had existed for one year, and had set in following a convulsive attack. At the onset, the vocabulary of the patient was reduced to a few intimate words, such as her name, the name of her husband, etc. To other questions she would answer: "I cannot." At other examinations she had difficulty in pronouncing words that she had previously spoken, but pronounced new words easily. She did not show disturbances in writing. She understood everything that was said to her, carried out simple as well as complicated orders well, and could read aloud.

Hysterical aphasia is distinguished from organic aphasia by the following: 1. There is an absence of agraphia in the former condition. 2. There is an irregularity in the aphasia—the person with organic aphasia pronounces only a few, and always the same words, while the person with hysterical aphasia has a richer vocabulary, pronounces new words, and often forgets words that had previously been pronounced. 3. The person with organic aphasia uses oligosyllabic words, and usually those that are emotionally toned (curse words, etc.), while the person with hysterical aphasia has no difficulty with polysyllabic words and those of intellectual content. 4. While the former tries to answer all questions with his few words and becomes impatient with his difficulty, the latter sits quietly and says, "I cannot." 5. The intelligence, at least in some particular sphere, is lowered in the person with organic aphasia; it is not in the hysterical person. 6. The person with hysterical aphasia has more difficulties in speech while she is being examined than when she is talking with other patients. 7. Sudden recovery following electrical stimulation speaks for hysteria.

IMPASTATO, New York.

CONGENITAL ABSENCE OF SWEAT GLANDS AND OZENA. O. FLEISCHMANN, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:503 (May) 1931.

Fleischmann first lists the cases of congenital absence of sweat glands combined with deficiency of hair and teeth, which he found in the literature. According to some authorities these cases form a special clinical group showing an ectodermal

defect of high degree, and they deserve special interest because the majority are combined with ozena. The relations of this disease to the malformation are as yet not fully explained. For this reason the author gives a detailed report of a case from his own observation. A comparison with the other cases reveals that the manifestations on the part of the skin can all be traced to an inhibition in its development by which an embryonal condition is retained, and the anlage of the cutaneous appendages is more or less suppressed. It can also be demonstrated that the same explanation applies to the entire mucous membrane developing from the stomodeum, because here one likewise finds an embryonal condition in the inhibition of the development of glands and teeth. In ozena concurring with a defect of the sweat glands a defect of the nasal mucous membrane is the cause of the ozena. Even in ozena that occurs as an isolated condition the same defect, although perhaps less pronounced, is probably the cause. This is proved not only by a study of the genealogical trees but also by the concurrence with other homogeneous manifestations, and likewise by the occasional simultaneous existence of other defects, particularly of the so-called ozena skull. As causes of the ectodermal defects neither congenital syphilis nor endocrine disorders were demonstrable.

EDITOR'S ABSTRACT.

RELATION OF SPEECH AND VOICE THERAPEUTICS TO OPERATIVE LARYNGORHINOLOGY. R. SOKOLOWSKY, Ztschr. f. Laryng., Rhin. (teil 1: Folia otolaryng.) **20**:537 (May) 1931.

In the introduction Sokolowsky states that he intends to show (1) when and what kind of operative interventions are necessary in disturbances of voice and speech, and (2) when and which operative interventions should be avoided. He stresses the second point since he intends to warn the practitioner against therapeutic mistakes that may lead to permanent disturbances of speech and voice. His report is based on several decades of practical experience. In the main part of the article he first discusses the relations of operative laryngology to the disturbances of speech. For disturbances of speech, such as retarded development of speech, idiopathic dumbness, stammering and stuttering, he gives as a rule that an operation on the upper respiratory passages is indicated only when the surgical procedure would be necessary in the absence of disturbances of speech. He considers division of the fraenum linguae ineffective for the correction of retarded development of speech as well as for idiopathic dumbness and for stammering. Adenotomy has been suggested as a remedy for stuttering. However, the author thinks that cases in which it is effective are an exception, and that, because the psychic shock might cause an exacerbation of the disorder, it should be avoided unless other conditions, such as inhibited nasal breathing or baryecoia of the middle ear, necessitate it. The rule about the inadvisability of operative treatment for disturbances of speech does not apply to all the forms of rhinolalia. In rhinolalia clausa organica surgical treatment is necessary. In rhinolalia clausa functionalis, however, an operation is superfluous and therefore contraindicated. In rhinolalia aperta in children plastic operation on the palate is advisable; in adults, an obturator. Other forms of surgical intervention on the nose and throat impair the speech more and are therefore contraindicated. Only in exceptional cases are they permissible. Special precaution is necessary in insufficiency of the velum palatinum (adenotomy should not be performed). In most cases of rhinolalia mixta, operative intervention on the nose and throat are contraindicated. Only in exceptional cases and under certain conditions are they permissible. The author further discusses the relations of operative laryngology to phonasthenia. In regard to operations on the nose, he says that if nasal respiration is inhibited the obstruction should be removed. In other cases only calm, careful deliberation and functional tests will prevent injurious interventions. Further he discusses operations in the nasopharyngeal cavity (adenoids), and operations on the pharynx (lingual tonsil, pharyngitis lateralis and palatine tonsils). The last part of the article treats of disturbances of the voice in organic diseases of the labia vocale.

In the discussion on the tumors, the author disregards neoplasms and says little about polypi and other tumors. So-called singers' nodules, however, are discussed in detail. For patients with singers' nodules the treatment should commence with enforced silence. However, if a spontaneous retrogression does not set in, operative treatment should be resorted to. Other forms of organic disturbances of the labia vocale which are discussed are the various types of paralysis. In unilateral paralysis of the recurrent nerve the author generally obtained good results with exercises and with other conservative methods, so that an operation was only rarely necessary. In discussing the treatment for paralysis of both musculi thyroarytaenoidei interni and of the musculus arytaenoideus transversus he describes an operation recommended by Katzenstein.

EDITOR'S ABSTRACT.

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY OTO-RHINO-LARYNGOLOGICAL SERVICES

EDITED BY PHILIP E. MELTZER, M.D.

Clinical Meeting, Jan. 22, 1931

HARRY A. BARNES, M.D., *Chairman*

A CASE OF MASTOIDITIS WITH ASEPTIC MENINGITIS. Presented by DR. CALVIN A. FAUNCE.

The patient, a boy of 15, well until two weeks before this presentation, developed acute rhinitis with right acute otitis media which ruptured spontaneously. The patient was without symptoms until a few days before admission to the hospital when he complained of severe pain in the right eye; his temperature was 105 F. For the next few days the temperature ranged from 103 to 105 F. at the time when he was admitted to the hospital by his physician with a diagnosis of lateral sinus infection. On admission there were no symptoms whatsoever, such as headache, chills, nausea or vomiting, but there was slight tenderness behind the ear with slight discharge from the middle ear. A lumbar puncture was done; the cerebrospinal fluid pressure was 180. A Tobey-Ayer test to determine the patency of the lateral sinus was negative. The patient appeared to be quite toxic, however. A simple mastoidectomy was done. All the cells were found to be filled with pus; the cell partitions were decalcified and beginning to break down. The mastoid process was large and deep. The lateral sinus and the dura were widely exposed. The cell count of the spinal fluid was 1,000, practically all mononuclears. The sugar content was 38 mg.

The patient improved steadily for about ten days, and the temperature returned to normal. The patient was seen by the members of the neurologic service, who agreed that the diagnosis of an irritable or aseptic meningitis was probably correct, and that the prognosis appeared favorable.

On the twelfth day, the patient suddenly complained of severe headache; there was a rapid rise of temperature to 104 F. and he became very restless, vomited and had rigidity of the neck. Lumbar puncture showed a cloudy fluid under moderate pressure; no organisms were recovered by smear. The condition of the patient became worse, and the cisterna magna was drained by suboccipital decompression and removal of the arches of the first and second vertebrae. This was done by the neurosurgical service in an attempt to get constant drainage. The neurosurgical service has operated in several such cases without good results, and this procedure is to be discontinued as far as the Eye and Ear Infirmary is concerned.

The interesting point about the case is that the patient probably had a localized meningitis in which there was a sudden invasion of the arachnoid space. Why he did not give evidence of the localized meningitis in the convalescent period is still unknown.

A CASE OF BRAIN ABSCESS WITH MENINGITIS. Presented by DR. P. E. MELTZER.

A woman, aged 45 years, had a simple mastoidectomy performed on the left ear sixteen years before presentation. She was perfectly well until July, 1930, when she had a severe earache for about two weeks, with pain and tenderness behind the ear, followed shortly by delirium and coma. A diagnosis of temporosphenoidal abscess was made by Dr. E. E. Jones, who explored the brain and evacuated the abscess. The abscess drained for seven weeks; finally the patient

was sent home from the hospital, improved. She was able to go about her household duties, complaining only of occasional headache. The postaural wound continued to discharge, and the patient noted that when the discharge lessened her headache recurred. She continued this way for a period of about six months, when she was admitted to the Eye and Ear Infirmary because of cessation of postaural discharge, severe headache, vomiting and drowsiness increasing to practically a comatose state. On admission the temperature was normal, there was no evidence of meningitis; the middle ear was dry and the postaural wound entirely closed over. Fourteen hours later, before the physician's usual morning house visit, all the signs of meningitis developed. Lumbar puncture showed a normal pressure, but there were 6,000 cells per cubic millimeter, with 97 per cent polymorphonuclears. Pneumococci were recovered from the smear of the spinal fluid. The brain was immediately explored through the previous wound, and a large abscess was again evacuated from the temporal lobe. Evidently the fistulous tract that led to the mastoid wound had closed over and the abscess cavity had refilled, giving the signs and symptoms described. At autopsy there was no evidence of generalized meningitis, but the patient did have a basal meningitis, and the pathologist found a tract from the abscess sac into the ventricle whereby the base was flooded with pus cells.

PROGRESS OF A CASE OF MASTOIDITIS. Presented by DR. C. R. JOHNSON.

A woman, aged 35, had a simple mastoid operation done in July, 1930. A month later a jugular ligation was performed, and the lateral sinus was investigated. In December, 1930, she came into the eye clinic with 3 diopters of choking in each eye, some unsteadiness on her feet, frontal headache and continuous discharge from the postaural wound. The temperature, pulse and respiration were normal. Roentgen examination showed all mastoid cells exenterated. Because of the postaural discharge and because the lumbar pressure was 300, the postaural wound was reopened, and a large pad of thick granulations was found over the surface of the dura of the middle fossa, which seemed to be under pressure in this region. A few days later, the temporal lobe was explored, but nothing was found. The lumbar pressure remains between 250 and 300. The fundi remain the same, but the patient's general condition is greatly improved. She is up and around and not unsteady on her feet.

DISCUSSION

DR. H. P. MOSHER: What is your diagnosis now?

DR. C. R. JOHNSON: The diagnosis is yet in doubt. The patient may have an arachnoid cyst formation or thrombosis extending backward to the torcula, interrupting the cerebral circulation and causing increased intercranial pressure.

DR. P. E. MELTZER: We have had several such cases in the past few years in which, following lateral sinus thrombosis, there was an increase in intercranial pressure as indicated by the monometer, choking of the disks, etc., without other intracranial signs or symptoms. These cases have been under observation for many years. One patient in particular had choking of the disk of 4 diopters, which has entirely cleared up; the disk has remained clear for two years.

A CASE OF TOTI-MOSHER OPERATION. Presented by DR. G. H. POIRIER.

A woman, aged 43, had complained of epiphora from the right eye for seven years when she was admitted to the eye service. It was impossible to pass a Bowman probe into the sac. There was no evidence of suppuration or of a mucocele. She was transferred to the nose and throat service. The roentgenologist reported left ethmoiditis. The usual Toti-Mosher operation was performed, the anterior ethmoidal cells and the posterior half of the lacrimal sac being removed. When the probe was passed from the canaliculus there was a definite obstruction so that it could not pass into the sac. The probe was forcefully pushed into this sac, and the constriction about the probe was cut with a Bowman knife. The external wound was closed in the usual manner. Each day for two weeks the canaliculus was dilated with a number 4 probe. Finally a Bowman

knife was inserted into the canaliculus and slit its entire length, so that a number 12 probe would pass easily. The canaliculus was probed daily with the number 12 instrument. There is practically no tearing at present.

DISCUSSION

DR. H. P. MOSHER: The tear sac operation as introduced by me originally has been modified considerably by Dr. G. Poirier and Dr. C. Porter, not in principle but in such minor details as you have heard in this case in which failure might occur if these little modifications were not made. The operation is continually being improved.

DR. G. H. POIRIER: It is reasonable to presume that 95 per cent of the cases we see are due to mucocele or to chronic infection of the tear sac. In most cases it is not necessary to bother with the canaliculus.

TOTI-MOSHER OPERATION. Presented by DR. G. H. POIRIER.

A boy 6 years of age was admitted to the hospital last November with an orbital abscess. A Killian incision was made to drain the abscess. No pus was found, and as roentgen examination showed no evidence of sinus infection, nothing more was done. A few days later, a fluctuation at the outer canthus developed. This was drained, and the youngster was sent home apparently well. A week later, he had cold in the head with profuse bilateral nasal discharge. The original incision became reddened and swollen. Roentgen examination showed that there was pus in both antrums. These were drained, and the child did well except for a continuously discharging fistula at the lower end of the incision. Iodized poppy-seed oil 40 per cent was injected into the right antrum, and it made its way into the tear sac. A Toti-Mosher operation on the tear sac was performed with excellent results. There was complete cessation of all discharge; the fistula is entirely healed, and the nose is free from secretion. I feel that when a Killian incision is used one should be careful in elevating the tissues away from the lacrimal bone in order not to traumatize the sac, for secondary infection is likely to occur with a chronic dacryocystitis.

TWO BRONCHOSCOPIC CASES. Presented by DR. D. C. SMYTH.

These cases showed the necessity of real study before attempting a bronchoscopy. The first case is that of a child from New Bedford, who was seen by a pediatrician. He had increasing dyspnea for two months. It is suggestive that two months before he was seen by the pediatrician he swallowed a button, that he chews gum considerably and, although he is 4 years old, is in the habit of swallowing the gum. Physical examination showed definite signs of obstruction in the trachea. The child was brought to this hospital late at night with a temperature of 104 F., and considerable dyspnea. The next morning he was taken to the fluoroscope room, and the anteroposterior view showed definite fusiform swelling just about the region of the great vessel. X-ray plates showed destruction of the bodies of the fifth and sixth dorsal vertebrae, with an enormous abscess. There was no external deformity in this case. The child had never had night crying.

The other patient came to me with a diagnosis of collapse of the lower lobe of the right lung. The history was that two months before the patient had a severe cold followed by a racking cough; no sputum had been raised, only a little mucus. The cough persisted for six weeks. One roentgenologist made a diagnosis of collapse of the lower lobe of the right lung because of a round shadow in the region of the right lower lobe. Further study of the case by Dr. A. S. MacMillan and myself showed that during respiration, when the diaphragm went down, the shadow extended upward. The patient was given barium by mouth, and a diaphragmatic hernia was easily diagnosed. I think that the diagnosis was entirely accidental, because the patient complained of this persistent cough, and an alert physician had a roentgen examination made of the chest.

A Case of Fibrosarcoma of the Pharynx. Presented by Dr. H. A. Barnes.

A youth, 17 years of age, came to the hospital in June, 1929, giving a history of dysphagia and dyspnea for one month. At that time he showed a large, smooth, hard tumor low down in the pharynx, which obstructed the glottis and caused the present symptoms. Dr. F. Garland removed the tumor by passing a snare around it, as it was pedunculated. It came out readily, and the base was treated with diathermy for seventeen months. The patient was then without symptoms.

The patient returned in November, 1930, because the symptoms reappeared, and on examination the condition was found to be the same as formerly. Dr. H. Tobey saw him this time, and repeated the same procedure. Recurrence was rapid, and he returned with a tumor fully as large as the one first described. Ten gold radium seeds were placed in the growth. It is now three weeks since this was done, and you will note, if you look in his larynx, that the growth is almost flush with the pharyngeal wall with slight sloughing over the surface. Radium had a very rapid effect on the size of this tumor. When all reaction has quieted down, it may be necessary to repeat the procedure with a smaller number of seeds.

DISCUSSION

Dr. T. E. Herman: I think it is just as well in a case of this sort to supplement the seeds with deep roentgen therapy.

Dr. H. Tobey: How soon do you begin roentgen therapy after removing the radium? Would you have operated or followed Dr. Barnes' procedure if you first saw the tumor?

Dr. Herman: I use the roentgen rays at the time of the implantation of the radium seeds. As to whether or not I would operate on the tumor, I generally like to remove the major portion of the growth and then implant radium in the base.

Dr. H. P. Mosher: Would a histologic section show whether it was useless to operate?

Dr. Herman: Biopsy is of great help, especially in the transitional types of tumor.

Plastic Operation for Congenital Cleft Palate. Presented by Dr. V. H. Kazanjian.

A man, aged 23, was born with a cleft palate and harelip. The lip was repaired shortly after birth; the palate unfortunately was not repaired, which caused marked palatal and facial deformity, as you can see from this plaster model. You will note the great distortion of the upper jaw, the teeth are massed together, and the occlusion is very poor.

The first step was to take out the teeth, and the second was to make a plate that caused the upper lip to be put on a stretch. This was done to increase the elasticity of the upper lip in preparation for plastic work in this region.

In the second operation the entire upper lip and nose were separated from the bone, and a skin graft was placed over the fresh surface. An artificial denture was then inserted to hold the graft in place. The irregularity of the nose was later corrected, and minor plastic work was done on the lip for symmetry.

Comparison of the model with the present appearance of the patient shows the tremendous change in facial expression.

DISCUSSION

Dr. H. P. Mosher: I have used up all my adjectives in praise for Dr. Kazanjian's work, and the patient has expressed his gratitude for Dr. Kazanjian's patience and effort with him.

A Case of Radical Obliterative Frontal Sinus. Presented by Dr. H. A. Barnes.

The patient complained of severe frontal headache over a period of seven years. The sinuses were large and shallow, and there were a number of partitions in

each sinus. A vertical incision was made in the midline of the forehead from the hair line to the root of the nose; a transverse incision was then made extending from the outer portion of one eyebrow across the root of the nose to the outer portion of the other eyebrow. The flaps were then dissected over the frontal bone and turned back, exposing the bone of the entire forehead. The bone over the sinuses was completely removed. The entire mucous membrane was markedly diseased and the sinuses were filled with purulent secretion. A bilateral ethmoidal exenteration was also done. The partition between the two frontal sinuses was removed down to the nasal septum. The soft tissue flaps were held back by sutures and the scalp, and the large cavity over the entire frontal bone was allowed to granulate in. This took several weeks, and then the flaps were replaced and the granulations were kept clean by packing lightly with hexylresorcinol solution S. T. 37 alternating with mercurochrome-220 soluble 4 per cent. There is considerable deformity present, and the patient is now to be readmitted for plastic operation. An obliterative frontal sinus was necessary because of the extreme size of the sinuses; the marked infection present caused an osteomyelitis of the front wall of the frontal bone, and there was no other procedure that would have eliminated this infection.

DISCUSSION

DR. G. TOBEY: Could you demonstrate by the roentgen rays that osteomyelitis was present?

DR. H. A. BARNES: Dr. A. S. MacMillan could definitely demonstrate osteomylitic areas.

DR. H. P. MOSHER: These operations are extremely radical, and I cannot quite agree with Dr. Barnes in the election of obliterative operation in all cases. I feel that one can get along without it.

DR. D. C. SMYTH: I think the time of primary obliterative operation on the frontal sinus has gone by, and we should use this measure only in cases in which several thorough operations have failed and the anatomy of the nose is such that it seems unlikely that the usual Killian or Lynch operation will succeed.

DR. BARNES: I feel that if one cannot thoroughly remove the bone partitions and the infected mucous membrane from remote recesses, an obliterative operation may be done. I do not do it in all cases as a primary operation. I agree with Dr. Smyth that in cases in which there have been repeated operations for relief of frontal sinusitis with recurrence, the obliterative operation is indicated. In this particular case a great deal of intranasal work had been previously attempted, without relief. The surgeon felt at the time that nothing but an obliterative operation would be of any use in such extensively developed and infected sinuses.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Feb. 2, 1931

GEORGE DENNIS, M.D., *President, in the Chair*

CARCINOMA OF THE LARYNX. DR. E. E. MEISTER, Aurora, Ill., and DR. ALFRED LEWY.

DR. E. E. MEISTER: A man, aged 57, was first seen on Nov. 19, 1930. He gave a history of hoarseness of five years' duration. In 1925, he was examined elsewhere; nothing abnormal was found in the larynx. He went to Arizona, but a roentgenogram of the chest and the results of tests with tuberculin were negative. The hoarseness persisted for about a year, when it cleared up somewhat; it always became more pronounced toward evening. He spent his winters in Arizona. In

the spring of 1930, he noticed a lump below the larynx, which increased to the size of a hen's egg. He had pain in the throat and difficulty in swallowing and breathing.

The patient came to Chicago to consult a goiter specialist, and a diagnosis of abscess of the thyroid gland was made. The abscess was opened, and serous pus was drained. He remained in the hospital for about four weeks, but a few weeks later the abscess was again opened, and the hoarseness became more pronounced. The difficulty in swallowing increased to the point where the patient could not take solid food.

When I saw him he was cyanotic and slept sitting up. He had not taken food for two weeks. Examination showed a mass in the midline of the larynx which obscured the vocal cords. He was sent to the hospital. Dr. Lewy was called in consultation, and a tracheotomy was performed. The patient lived for about five weeks, although breathing was very uncomfortable, and he was unable to swallow food.

The Wassermann reaction was repeatedly negative.

Dr. Alfred Lewy: When I first saw the patient, he had a large suppurating swelling that occupied most of the front of the neck. I made a provisional diagnosis of carcinoma of the larynx, and an immediate tracheotomy was performed as he was in a critical condition of anoxemia.

The history stated that in April, 1930, an incision had been made in the swelling, and that the patient was subsequently treated for syphilis by a well known physician. The tracheotomy was performed under local anesthesia, with the patient sitting up; the thyroid had to be opened in order to place the tube.

Examination of a pathologic specimen showed squamous cell carcinoma.

DISCUSSION

Dr. Harry L. Pollock: It is possible that a carcinoma may have existed as long as the tumor in this case. About a month ago I operated on a patient who had first noticed hoarseness seven years before. It persisted for two years in the beginning, and then had practically disappeared for a short time; however, it recurred. The condition was a slowly growing carcinoma. We have always tried to point out that hoarseness that improves at times but never disappears, excluding tuberculosis and syphilis, is almost pathognomonic of carcinoma.

Progressive Osteomyelitis of the Cranial Bones Secondary to Suppurative Nasal Sinus Disease. Dr. C. F. Yerger.

There are two types of progressive osteomyelitis due to disease of the nasal sinuses; a type that develops spontaneously and a postoperative type. The frontal sinus and the frontal bone are most frequently involved. In the majority of cases, the causative organism is the staphylococcus. The pathologic process is characterized by progressive osteomyelitis of the frontal bone or of other cranial bones. The spread may be by continuity of the diseased bone or by involvement of the healthy bone via infective thrombophlebitis of the diploetic veins. Besides the osteomyelitis, two other pathologic conditions are associated, the pericranial or subperiosteal abscess and the extradural abscess. Clinically, the pericranial abscess is readily recognized by the formation of a soft, doughy or puffy swelling over the site of the osteomyelitis. This was first noted by Pervical Potts more than 170 years ago in cases of osteomyelitis of the cranial bones due to external trauma. The presence or absence of an extradural abscess in these cases is not so easily established, and this is the great hidden danger of the disease.

In order to establish the etiology of disease of the nasal sinuses, an organism must be demonstrated as the causative factor. This was first accomplished by Luc and Tilley, in 1899; since then about 100 cases have been reported. The roentgenogram is an important aid in diagnosis and in obtaining a graphic chronological record of the course of the disease. Patients with these cases are

prone to have relapses, and no patient should be considered cured until after the lapse of at least one year.

The prognosis is unfavorable. McKenzie noted that the mortality in post-operative cases was 70 per cent, and in spontaneous cases, 30 per cent. Cases in which there are intracranial complications, such as meningitis, abscess of the brain and thrombophlebitis of the dural sinuses, are fatal.

The surgical treatment for pericranial and extradural abscesses should be based on wide exposure and a wide open wound with early drainage. All loose sequestrums should be removed, and the wound should be packed with petrolatum gauze. The bone should be allowed to sequestrate. It should always be kept in mind that an intact lamina interna may hide an extradural abscess. As much of the internal table should be saved as is possible, as the bone regenerates from it. The anterior and posterior wall of the frontal sinus should be removed, but the supra-orbital ridge and the inferior wall of the frontal sinus should be conserved.

DISCUSSION

DR. HARRY L. POLLOCK: I wish to emphasize one or two points. Dr. Yerger stated that in cases of scarlet fever, influenza and so on there is always a lowered resistance. That is true of all these diseases, and I think that it is to be expected as the body has not had time to build up an immunity. He also said that the patients are treated until they build up an immunity, but the acute infections come on so abruptly that there is no time to build up the resistance.

I believe in early radical treatment in these cases. They are comparatively rarely seen in private practice, but I have seen some. I think that many of the 100 cases reported were not seen by rhinologists. I wish to mention 2 cases in which the patients were under the care of general surgeons when I was called in consultation. The first patient had a typical soft swelling, a puffy tumor, which the surgeon opened and curetted. Soon a similar spot appeared on the opposite side, for which the same treatment was used. I agree with Dr. Yerger regarding too early operation, but I think that radical measures must occasionaly be used when there is involvement of the ethmoid, frontal or any other sinus. I believe that many of the complications are due to too early operation, before nature is given a chance to wall off the pathologic process. In the case mentioned, I advised an extensive operation; the tumor was entered as far beyond the border of the sequestrum as was possible, in order to remove the diseased bone. It is far better surgically to take away too much than too little bone, for if the removal is not carried into healthy bone, the disease will continue and progress until the whole cranium is involved.

I wish to present a slide that demonstrates (1) the great amount of bone that can be removed from the cranium without any untoward symptoms and without any great amount of deformity and (2) the amount of regeneration that occurs in the formation of new bone, both from the edges of the old healthy bone and from the outer layer of the dura. The slide is an x-ray picture of a young woman who had osteomyelitis of the skull following an operation on the mastoid. She was under the care of a general surgeon. At least a dozen spots may be seen over both sides of the skull, from which large amounts of bone were removed at various times for a period of years. The patient finally made a complete recovery; i. e., she has remained free from symptoms for more than two years. The areas that were operated on early now show the formation of new bone; if examined closely, one can tell by the amount of new bone just where the disease began, and its progress can be followed. I saw the patient last summer, and on physical examination of the skull, I could scarcely detect any irregularities, demonstrating that regeneration had occurred over the entire operative terrain.

DR. AUSTIN A. HAYDEN: I wish to call attention to the fact that in the January, 1931, issue of *Laryngoscope* there is a report of an operation on the frontal sinus, with recovery.

DR. EDWIN MCGINNIS: I have had seven or eight patients with osteomyelitis about the frontal bone at the Presbyterian Hospital whom I have treated surgically,

with no deaths. There are hundreds of persons suffering from infections of sinuses who do not have osteomyelitis. Many patients have been operated on externally, and *Staphylococcus aureus-hemolyticus* has been found in the discharge, without osteomyelitis. I think that the important factor in the etiology is that *Staphylococcus aureus-pyogenes* is brought into the wound as a contamination, and this produces the osteomyelitis. When I first read LeMere's report of cases that resulted in death, and then heard Lillie, and later Ross Skillern, tell the same sad story, I was very much "down in the dumps." However, at the sectional meeting of the American Medical Association at Portland, Ore., in 1929, Dr. Samuel Skillern reported the case of patient he had treated, which resulted in a cure. One evening many years ago, I listened to Dr. Phemister talk about osteomyelitis of the long bones, and I learned a lesson in the treatment of patients with this condition. I have made wide exposures through long incisions, removed the sequestrums, if any were present, and then packed the wound with iodoform gauze. This packing is left for a month or six weeks, or until all of the advancing infection has stopped. It is with this method that I have been able to obtain cures in patients with this difficult condition.

DR. JOSEPH C. BECK: I wish to report the case of a child with acute sinusitis whom I saw in consultation with Dr. Woodruff in Joliet. An incision had already been made over the infected sinus. The child recovered from the acute condition. I saw him about a year later with an infection in the arm. A general surgeon to whom I referred the case made a diagnosis of secondary infection of osteomyelitis of the humerus. At about the same time, a puffy tumor developed over the left side of the head. The resistance and general condition of the boy were very good. Two general practitioners saw the case in consultation, and one advised radical operation on the head and the arm, while the other one and I urged conservative treatment. I mention this fact because of the use of the LaPorte treatment, which consists of the application of a mixture of potassium nitrate and oatmeal. After I had made an incision and drained the tumor of the head, we used the mixture as a poultice. Dr. A. S. Strauss used the same treatment on the arm. Daily dressing and the application of the LaPorte mixture were carried out. The boy has recovered full function in the arm, and the swelling of the head has disappeared. A roentgen study of both the head and the arm has been most instructive. At one time the clinical picture as well as the roentgen appearance of the skull indicated progress of the disease, and I seriously thought of performing the extensive operation recommended by Dr. Furstenberg of Ann Arbor. However, the process again subsided and now appears to be cured. I have seen several cases of this type, in one of which I performed an exposure of the frontal sinuses in the belief that the condition was bilateral sinusitis. I found that it was late osteomyelitis, and took off the whole outer table of the frontal bone.

In the early stages of osteomyelitis, the operation is not well tolerated. Dr. Kemp, of Los Angeles, showed me a case of acute osteomyelitis in which he had reached the bone entirely across the head (from ear to ear) through the skin by means of an electrocoagulation knife; the patient recovered. The purpose of the electrocoagulation was to stop the progress of the pathologic process. Dr. Lynch, in his operations on many frontal sinuses, has shown that by great care in lifting off the periosteum from the frontal bone, infection of the bone has been prevented. I am sure that Dr. Yerger is right in his statement that many wounds, particularly in the bone, become infected at the time of operation.

DR. GEORGE H. MUNDT: Much might be learned from the work of Dr. Orr, of Lincoln, Neb., and of other orthopedists regarding the treatment for these infections: to leave the wound open, to pack with petrolatum gauze and to leave it there for weeks or months. Using Orr's method, one investigator in Chicago has done even more work than Dr. Orr, and another in New York has done considerably more work. The practice of applying iodoform gauze, pulling it out and repacking the wounds from time to time has its disadvantages.

DR. C. F. YERGER: I wish again to stress the point mentioned by Dr. Lambert, that it is important to remove the internal plate of the frontal bone for the purpose of either locating or excluding the presence of an extradural abscess, which is the great danger in cases of progressive osteomyelitis of the frontal bone.

I was glad to hear of the vast experience and the splendid results obtained in the treatment for progressive osteomyelitis of the frontal and other cranial bones secondary to disease of the accessory nasal sinuses as related by Dr. McGinnis. His cases offer additional proof that a suture should not be made in such cases, and that the wound must be left wide open.

In order to avoid the possibility of secondary osteomyelitis as a complication following a Killian operation on the frontal sinus, one should be careful not to detach any more of the periosteum over the frontal bone than is necessary and to have the bone beveled so that there will be no overhanging edges with an undermining of the posterior bony margin. One should also be sure to provide good intranasal drainage by means of a rubber tube.

EXTERNAL OPERATIONS ON THE FRONTAL SINUS. DR. AUSTIN A. HAYDEN.

(Dr. Hayden presented motion pictures of patients on whom he had operated.)

DISCUSSION

DR. J. HOLINGER: Dr. Hayden's motion picture demonstration was instructive. The first patient, who had an enormously swollen upper eyelid that hung over the lower lid, came to Dr. Blomgren, an assistant of Dr. Hayden, during his absence. I operated on the patient as an emergency, since it was evident that he had a retrobulbar abscess. He had experienced several lighter attacks previously. There was no secretion in the nose, and a probe did not enter the frontal sinus. A roentgenogram showed a large cloudy sinus; the limits were blurred. When the sinus was opened, the periosteum could only be freed from the bone with difficulty. Tough connective tissue was encountered everywhere, in which the trachealis muscle was enveloped (old scars from former inflammations). About 1.5 cm. from the supra-orbital arch, an abscess containing 30 cc. of pus under pressure was opened. An opening in the roof of the orbit, 2.5 by 1 cm., led into a large frontal sinus; from this sinus another opening in the depth led into the extradural space. A communication with the nose could not be found. Therefore, the tumor was an empyema, or an extradural abscess, of the frontal sinus which had no communication with the nose, but a fistula into the orbit and another into the skull cavity.

In the "Yearbook for Eye, Ear, Nose and Throat" for 1930, the following statement is made:

"In this country there springs up from time to time the advocates of radical and external operations. The reason for this seems to be that these men who are advocating operations of this sort have not had the patience to master the difficult intranasal surgery required to carry out a successful operation and their preparation has been more along the line of a smattering of general surgery which they proceed to apply to the external operation and apparently without any definite realization of what the proper indications on the sinus really are."

It is unfortunate but true that specialists in diseases of the ears, nose and throat work out the special anatomy, special diagnoses, special indications and special technic for the actions in their territory. Being ignorant of this work, some surgeons invade this territory. Many of these invasions are not for the best interest and welfare of the patients.

DR. C. F. YERGER: Dr. Hayden's motion picture demonstration and his explicit explanatory legends have been instructive. He is to be congratulated for his photography.

The fact that progressive osteomyelitis may follow a radical Killian operation on the frontal sinus, because this operation frequently requires opening of the

diploetic spaces in the frontal bone and the detachment of the periosteum over the frontal plate, would lead one away from the Killiam to the Lynch operation, as a means of lessening the danger of this complication.

DR. EDWIN MCGINNIS: I was glad to see Dr. Hayden's pictures and to find that he used the simple Killiam procedure in his work. I have more often used the modified Lothrop operation in attacking the frontal sinus externally; however, both operations are equally good in treating the infection.

Regular Meeting, March 2, 1931

GEORGE DENNIS, M.D., *President, in the Chair*

AGRANULOCYTIC ANGINA. DR. FRANK J. NOVAK, JR.

My report is based on a study of seven cases of agranulocytic angina that occurred in my practice. Six of the patients died, and one recovered. Owing to the multiplicity of therapeutic agents employed in the desperate effort to save the patient's life in the case in which recovery occurred, a critical appraisal of any single agent is impossible. However, it was noted that during the six weeks of the patient's illness, the most definite rise in the number of polymorphonuclear cells occurred when a nonspecific protein (Omnadin) was being injected. This increase in cells, however, was not permanent. Following the injections of protein, roentgen therapy was employed, and it seemed that the permanent improvement in the blood picture took place then.

If the essential pathologic change in this disease is a noninflammatory liquefaction of the bone marrow of such bones as the sternum and the ends of the ribs, it is difficult to see how stimulating doses of x-rays or the injection of nonspecific protein can stimulate myelopoiesis. One may speculate on the possibility of an extramedullary myelopoiesis. That this does occur in other conditions, for instance, following frequent and severe bleeding of experimental animals, is an accepted fact. On this basis it is possible that in the case of the patient who recovered not all of the marrow concerned in the formation of granulocytes underwent necrosis. The stimulating effect of the injections of protein plus that of the x-rays forced the remaining available marrow to supply the organism with new granulocytes. Other organs not normally given to the elaboration of polymorphonuclear neutrophils, such as the walls of large blood vessels, were stimulated to an unusual activity.

Two of the seven cases reported were not diagnosed ante mortem. For many months, one patient had undergone a long series of injections in the treatment for acute arthritis. I was unable to learn the nature of this treatment. In this case, the patient died five days after the onset of angina. One patient died of hemorrhage from the throat. Three of the patients were men, three women and one a girl, aged 12.

In all of the cases, a smear taken from the ulcers showed *Spirillum* and a fusiform bacillus. The course was febrile to the end except in the case of the patient who recovered; he had a normal temperature after the first week of the illness.

All of the patients had ulceration of the throat. The ulcers were multiple and steadily increased in size, except in the case of the patient who recovered. In that case the ulceration disappeared after six weeks.

Until the essential pathologic process is worked out and the etiology becomes known, it is highly improbable that a satisfactory therapy will be developed. In the meantime, the clinician is forced by the malignancy of the disease to attempt any and all forms of therapy that have been employed.

DISCUSSION

DR. J. P. SIMONDS: From what I have seen of the pathologic changes in this condition, it seems that the bone marrow is disabled so far as the production of polymorphonuclear leukocytes is concerned. In the cases I studied there were areas of necrosis that involved all of the cells in the bone marrow indiscriminately in the necrotic foci. In the same cases, however, there was evidence of the formation of red blood cells, (sometimes a ring occurred around the area of necrosis) but polymorphonuclear leukocytes were not noted. What it is that has such specific action on the cells that produce polymorphonuclear leukocytes in the bone marrow I cannot say. Whatever it is, it seems to be quite specific, and yet this specificness overflows enough to involve all parts of the bone marrow in some parts of the structure. I do not know of any drug, bacterical toxin or physical phenomenon that has such a specific action. The effect of benzene is, perhaps, the nearest approach to it.

I think that after thoroughgoing thinking along the lines of the possibilities in the case and the nature of the formation of leukocytes, a promising line of investigation would be to try to find a substance that would have a specific effect on bone marrow. Ehrlich investigated 606 chemical substances before he found the one specific for syphilis. It may be that some one with equal patience and persistence might find the causative chemical substance that is specific for this condition. Whether or not the investigation led to the discovery of a specific substance for the treatment for agranulocytosis, it would open a field of research that would be extremely interesting.

The pathologist feels that the underlying pathologic process of every disease furnishes the only rational basis for treatment. As long as only the effects of angina and not the agent that produces it are known, it is not known how to combat the disease. It seems to me that any treatment that gives any possible hope, no matter how empiric it may be, is worth trying. Some years ago nucleic acid was used to prevent peritonitis, because it was believed to stimulate the production of polymorphonuclear leukocytes. Any substance that will stimulate the formation of bone marrow should be of value, provided there is enough substance left in the bone marrow to be stimulated. I have seen references to the use of serum from patients who have recovered from the disease, and I wonder if any one here has used such a method. It seems that it might offer a possible hope of good results. If the organism is ever found, if the condition is due to bacterial infection, it may be possible to produce an antibody that will act as a therapeutic agent in this condition.

DR. LAURENCE HINES: I have had the opportunity of making postmortem examinations and of observing the condition clinically in three patients in the last few months.

One point I might mention is the frequency of degeneration of the liver and jaundice. In the three patients I saw there was marked jaundice, and the liver showed pronounced necrosis without inflammatory reaction. In one case, a culture taken from the ulcerated area was positive for diphtheria bacilli. This produced a confusing clinical picture. Diphtheria antitoxin did not cause improvement.

Another factor that impressed me was the infrequency with which Vincent's *Spirillum* was found, in spite of the fact that the disease originally was supposed to be spirochetal. I brought with me specimens from two patients. One case showed involvement of the larynx, pharynx and trachea, with typical ulceration; the other case did not present pharyngeal ulceration, but had typical lesions in the esophagus and colon. There are two specimens from the colon, one showing widespread areas of necrosis and the other localized necrotic lesions. In the esophagus of this patient there were localized areas of necrosis. She had dysphagia, which was a striking symptom throughout the illness.

DR. CHARLES B. YOUNGER: I have the distinction of having been associated with a patient with agranulocytic angina who recovered. I do not claim that I cured the patient. The case was seen a year and one-half ago, and the patient is living and well, so far as we know.

I had occasion at that time to look up the literature on the subject and was impressed by the lack of uniformity in the reports of these cases. Some were called agranulocytic angina and others agranulocytosis, and only a few corresponded with the pictures of the first two cases reported in Europe. Some of the patients did not present involvement of the throat, and yet there was a great diminution of leukocytes and polymorphonuclears.

The case I was connected with occurred in a man who was then 68 years of age. He had been vigorous all his life, and did not give a history of illness. He became ill with what his family physician thought was a peculiar tonsillitis of follicular type. I wish to emphasize the finding in this case of a phenomenon that I believe exists generally in this type of case, and that is marked prostration. The patient was about for four or five days, gradually becoming weaker. Cultures of specimens taken from the throat showed a great preponderance of staphylococci. There were ulcers around the teeth and one in the right tonsil which became larger. The typical Vincent's bacillus and *Spirillum* were recovered from these ulcers. We did not succeed in finding *B. pyocyaneus* on examination of the blood. When I first saw the patient, attention had just been called to agranulocytic angina; because the condition in the throat and the other findings did not conform to any of the conditions of the throat with which I was familiar and because I was interested in this rather new subject, I thought that it might be that disorder. I had the patient taken to one of the hospitals with instructions to have a blood count made. A short time later the technician called me and said that there were less than 1,000 leukocytes and no polymorphonuclears. The next morning the blood was typed, and the patient was given a transfusion. He received three transfusions within a week, at the end of which time he was improving. There was a slight increase in the polymorphonuclears following the transfusions and a gradual improvement in the general condition. The patient was discharged in about two weeks. In addition to the transfusions, he was given large doses of quinine.

DR. GEORGE T. JORDAN: We saw an interesting case at Mercy Hospital in a patient who recovered. He was a patient of Dr. Sweeney, who I am sure will be glad to tell us about the case.

DR. LEO P. A. SWEENEY: The patient was a priest, aged 34. I first saw him on a Monday, when there were small hemorrhages over and between the left lateral and central incisors. He was sent to the hospital in order to have a count of the white blood cells made. On Tuesday, a blood count showed 950 leukocytes, with no polymorphonuclears, or transitionals and 66 per cent of lymphocytes. The lesions had spread behind the same teeth on the left side of the mouth. On Wednesday, the white cell count was 1,025, with no polymorphonuclears or transitionals. On Tuesday and Wednesday, he was given 0.45 Gm. of neoarsphenamine and roentgen treatment. On Thursday, the white cell count was 1,350, with no polymorphonuclears or transitionals. He was given another roentgen treatment and, in addition, subcutaneous injections of whole blood into the hip and of turpentine into the thigh. The mouth and throat were very sore, and sometimes it was necessary to give the patient as much as three-fourths grain (0.048 Gm.) of morphine at a dose. On Friday, the white cell count was 1,100, with 2 per cent polymorphonuclears and no transitionals. He was given another roentgen treatment and another injection of leukocytic extract. On Saturday, the white cells had increased to 1,150 with 4 per cent polymorphonuclears and 16 per cent transitionals. The hip was sore. The lymphocyte count was 80 per cent; the day before it had been 92 per cent. He was again given roentgen therapy and injections of blood into the thigh. On Sunday, a blood count showed 1,300 white cells, with 3 per cent polymorphonuclears and 34 per cent transitionals. He was given another injection of leukocytic extract; the thigh was very sore.

On Monday, a blood count showed 4,500 white cells, with 46 per cent polymorphonuclears, and 46 per cent lymphocytes; leukocytic extract was given. The thigh was extremely sore, red and swollen.

On Tuesday, the blood contained 6,700 white cells, 35 per cent polymorphonuclears, 35 per cent transitionals and 23 per cent lymphocytes. The temperature was 99.6 F.

On Wednesday, there were 7,950 white cells, 50 per cent polymorphonuclears, 12 per cent lymphocytes and 33 per cent transitionals. The number of lymphocytes remained between 20 and 30 per cent. There was evidence of a true abscess in the thigh, which was very sore. The mucous membrane was hanging down in the patient's monuth; we clipped it off in several places so that it would not trouble him. The temperature was normal, and remained so. The pulse rate was under 100 and a week later, under 90.

On Thursday, the white cell count was 8,050, with 54 per cent polymorphonuclears; on Friday, 8,100, with 55 per cent polymorphonuclears.

On the following Tuesday, the fourteenth day in the hospital, there were 7,750 white cells, with 62 per cent polymorphonuclears. On Friday, the seventeenth day, the white cells numbered 7,050, with 67 per cent polymorphonuclears. On Wednesday, the twenty-second day, there were 7,200 white cells, with 63 per cent polymorphonuclears.

Four weeks after the diagnosis had been made, the blood contained 7,150 white cells, with 58 per cent polymorphonuclears; after five weeks, 6,900 white cells, with 59 per cent polymorphonuclears, and after six weeks, 9,400 white cells, with 69 per cent polymorphonuclears.

The temperature ranged between 101 and 103.4 F. until about thirty hours after the white cells began to increase. The pulse rate was high at all times, from 120 to 124.

The patient is apparently in good condition at present. The two incisors over which the lesions first appeared dropped out while he was still in the hospital, and he has since had the two molars on that side extracted. It is now eight weeks since the onset.

DR. FRANCIS L. LEDERER: During the last ten years I have seen fourteen cases of this disease. The treatment is hit-or-miss. There is nothing about the therapy to which importance can be attached, and so we are of the opinion that the patients get well despite the treatment. In our series there have been two recoveries. The jaundice, as Dr. Hines mentioned, gives a poor prognosis. We are of the opinion that the angina is not a part of the agranulocytosis, but is a manifestation of the severe toxemia. We have seen two cases recently that ended fatally. We were able to observe the lesions early; they were hemorrhagic at first, with marked demarcation of the hemorrhagic area, and then gangrenous sloughing occurred. In one case, we thought that if we could get rid of the toxic area we could eliminate the toxemia; following the removal of the slough, the patient improved and recovered. The outstanding factor is that we, as laryngologists, have been prone to use arsphenamine in cases that resemble Vincent's angina. On looking through the literature, we are forced to wonder whether or not the arsphenamine precipitated the trouble. We have been watching and trying to follow out the careful use of arsphenamine in the treatment for Vincent's angina, for we feel that we might be instrumental in precipitating this type of angina. We remove the membrane very gently by evulsion.

DR. AUSTIN A. HAYDEN: I do not know how many cases of agranulocytic angina I have seen during the last nine years, but in the last eighteen months I have seen eight cases. I think that we have all seen more cases than we realize. To the time Schultz published his first paper, the last thing a specialist in diseases of the nose and throat or a general practitioner thought of in the care of a patient with any sort of ulcerative stomatitis was making a blood count. He made a culture immediately, but not a blood count. This condition has made evident the necessity of making blood counts, especially of the white blood cells, in cases in which there are ulcerative lesions of the nose and throat, especially of the throat. Seven of the eight patients I have seen are dead. I saw the only one who is alive in consultation with Dr. Frank Novak, and there was some question as to whether the case was really agranulocytic angina.

That brings up the question as to which cases should be classed as this disorder. How low must the white cell count be before one encounters the resistance offered by the white cells? In our series, all of the patients who died had a white cell count lower than 500. I think that when the white cell count is lower than 5,000, the prognosis needs to be guarded; when it is lower than 1,000, the prognosis is unfavorable, and when it is lower than 500, the prognosis is extremely grave. In the eight cases, there were two men and six women, all past middle life. The patient whose case is most vivid in my mind is the wife of one of the members of the staff at St. Joseph's Hospital. I shall give the history briefly.

About four weeks before angina developed in this patient, she had had a tooth removed; her husband was much incensed at the dentist because he sewed up the cavity. The wound healed without much trouble, but some soreness remained. In the morning, the husband went to the country, after he had called me to look at his wife's throat. She seemed to be perfectly well. Whether or not the blood count was increased or diminished at that time I do not know, but in view of the fact that blood counts change rapidly when taken four or five times a day, I suspect that it showed little change. Her husband suspected that she had an involvement of the antrum, and I was asked to examine her. Instead of an involvement of the antrum, I found a well marked membrane on the left tonsil. Culture showed Klebs-Löffler bacilli. Instead of healing the abscess persisted. A blood count showed less than 500 white cells. Within forty-eight hours, the patient was dead, in spite of the fact that she was given two transfusions of blood and enormous doses of diphtheria antitoxin.

To sum up, I think that the facts that stand out in this condition are the prevalence of syphilis, the rapidity with which the white blood cells increase and decrease and the occasional history of infection of the teeth. When I first came to Chicago I shared an office with an extraction specialist, and I know that some of his patients died of an infection of the throat. Whether or not they had agranulocytic angina I do not know, but a blood count would have been in order.

DR. ALFRED LEWY: I wish to report a case in which the patient recovered. A woman of middle age had what appeared to be a peritonsillar abscess. Examination of the blood showed the white cell count to be as low as 2,100, with less than 7 per cent granulocytes. The condition remained the same for a week or two, the granular leukocytes never exceeding 7 per cent, and sometimes going as low as 3 per cent; she then recovered. Three months later she had another attack of what appeared to be a peritonsillar abscess, with the same observations on the blood, and she again recovered. The only treatment she received was roentgen therapy in suberythema doses.

Some months ago I saw a report of twenty-six or twenty-eight cases of agranulocytosis in which one-twentieth suberythema doses of roentgen rays were given, with approximately 50 per cent recoveries. In our case we used one third of an erythema dose.

I reported a case of agranulocytosis with death before this Society three or four years ago, in which a blood culture revealed *Streptococcus viridans*. This organism did not appear for over a week after the culture was planted, and I do not know whether it was a contamination.

I think that the roentgenotherapy should be considered in these cases. Also, particularly in cases in which hemorrhage occurs, other cells besides the granular leukocytes may be affected, according to the degree in which the cell-forming portion of the bone and marrow is affected. There may be complete destruction of the bone marrow, as occurred in a case I saw recently in which hemorrhages occurred in the mucous membrane and other parts of the body. The case was diagnosed as panmyelophthisis. At postmortem examination, the medullary portion of the long bone was found to be about the diameter of a lead pencil.

DR. FRANK J. NOVAK, JR.: From the reports we have heard it is evident that many therapeutic measures have been employed in these cases and that it is difficult to state definitely that any one accounts for the improvement in some of the cases. Roentgenologists differ as to whether there is a stimulating dose of

x-rays. Among the patients to whom I referred there were one child, aged 4, four women and two men. Fusiform bacilli and Vincent's *Spirillum* were found in each case. In the case Dr. Sweeney mentioned the abscess seemed to have some effect on the progress of the case. Extensive necrosis of the bone marrow seems to be the underlying pathologic process in agranulocytosis, but it is not known what causes the necrosis. It is an interesting fact that while myelocytic elements are formed in the marrow, nevertheless under certain pathologic conditions, such as in repeated bleeding of animals and following the use of various chemical agents, these cells are also formed elsewhere. They are found in the aorta, the lymph nodes and even the liver. The process is that of acute extramedullary myelopoiesis. If the blood-forming elements are destroyed, perhaps the formation of the abscess in Dr. Sweeney's case and the use of turpentine stimulated production of the granulocytes by other organs until the bone marrow was able to take care of the situation. That explanation, however, is merely speculative.

Dr. Hayden mentioned the rapidity of the appearance and disappearance of granulocytes in the blood and the rise and fall of the total count. In one of our patients who had 1,100 white cells, 28,000 white cells, with 80 per cent granulocytes, developed within six hours.

DR. T. C. GALLOWAY: Our patient had necrotic lesions over the alveolar ridge, the tonsils and the tongue. There was a lesion over the arytenoids and the aryepiglottic folds which was not seen until the first ones began to clear. When a change in the blood picture occurred, all of the other lesions except this one began to slough and clear up. We watched the patient carefully and gave her inhalations of oxygen, but it became imperative to perform a tracheotomy. The wound is now clean, and there is no sign of the gangrenous condition that was present in the other lesions.

DR. J. P. SIMONDS: There is one possible way of throwing light on the etiology. Some patients have recovered. If the infection is really bacterial, the blood from these patients may contain agglutinins. It would be of value to take blood from the convalescent patients and to make agglutination tests on every organism recovered from patients with the disease. In that way we may be able to learn something of value for future use.

INDICATIONS FOR OPERATION IN ACUTE MASTOIDITIS. DR. A. H. ANDREWS.

(Dr. Andrews gave the indications for operation in acute mastoiditis.)

DISCUSSION

DR. FRANCIS L. LEDERER: I think that it is agreed that each case is an entity; a direct set of rules cannot be formulated to cover all cases. It is often purely personal experience that guides us in the management of an individual case and makes surgical intervention desirable. Two factors should be borne in mind: (1) the safety of the patient and (2) the future of the patient, particularly regarding hearing, prevention of chronicity and the like. We have all heard of severe cases in which recovery occurred without treatment, and it is therefore difficult to judge operative indications or intervention by medical management based on those cases. As long as an actual principle cannot be set up, I think that, as in every other disease, we should never forget that the underlying pathologic state is the determining factor in governing the management in these cases. We recognize that indications for operative intervention are often dependent on the ability and the temperament of the operator. We have always considered our indications under three groups: absolute, relative and atypical.

It should be realized that there are seasonal, individual, bacteriologic, anatomic and yearly variations in the symptoms in these cases. A case that is seen this year will not be the same as one seen last year. Much depends on whether or not we are dealing with an encapsulated organism, and we must consider the individual patient in every instance.

In the anatomic consideration, it is unnecessary to have a roentgenogram made in the early stage to aid the pathologic interpretation, except that it is well to know whether the case is one in which an early breaking down (rheumatic) or a greater local resistance (diploeic) can be expected.

In regard to early diagnosis, I think that tenderness at the onset of otitis has provoked more discussion than any other symptom, but I am sure it will be agreed that that symptom is periostitis and is not an indication for operation. We differentiate between mastoidism, which is periostitis, and empyema, which is really mastoiditis. The roentgenograms show variations and must be interpreted with a great deal of common sense. The clinical observation should be considered before the laboratory observations. The finding of a high calcium content in the pus has been considered an indication of the amount of destruction of bone, and in some instances it has served to aid in the making of the decision to operate. Wittmaack thought that when otitis media occurs with involvement of the mastoid before the age of puberty it interferes with the pneumatization of the mastoid. I think that this theory has been disproved. In the roentgen examination, we always take into consideration the question as to whether the patient has had a previous attack that might have altered the arrangement of the cells.

DR. GEORGE H. MUNDT: The essential factor, as Dr. Andrews and Dr. Lederer brought out, is to visualize what is happening in the mastoid, for the decision as to whether a case is operable depends on the condition of the mastoid. I believe that roentgenograms that are made after a few days and in a series are valuable. This procedure does not necessitate a great expense.

If one can feel bone in the débris that is discharged, this also is a valuable finding. We must certainly take into consideration the general condition of the patient. We have probably all seen patients with acute suppurative otitis media who were prostrated almost immediately. Many of these patients have a tremendous discharge from the ear. If this occurs and if the general condition is good, I think that it is thoroughly justifiable to open the mastoid early, although I think that it is undesirable to operate early in most cases. If the discharge is so profuse that one finds it difficult to believe that it comes from the middle ear, operation is necessary. In new cases of otitis media, one must decide the period at which one will begin to think seriously of the necessity of operating. For a good many years, I have become much disturbed over ordinary cases under good management in which the condition does not begin to clear up at the end of two or three weeks. I appreciate that the time element is unscientific, but I expect there is not a man present who does not consider it. Dr. Andrews spoke of deafness as a symptom in determining this point. Certainly in the cases of patients who become very deaf something should be done. I have never looked for the bony débris microscopically. I think that the soft tissue change around the tip of the mastoid, referred to by Dr. Andrews, is significant. It has been my practice for many years to ask patients not to tell which ear is involved, for I like to examine for myself, and I think that I obtain much valuable information in this way.

I have had experiences that made me dubious as to the justifiability of spinal punctures when these patients have meningism. I think that we can change meningism into meningitis without much difficulty. If the intracellular septums are broken down, it is just as well to operate at once, in about three weeks, whether this condition is found by means of a roentgenogram or by other measures.

DR. JOSEPH C. BECK: I have not operated on an acutely infected mastoid for some time, but I think that tomorrow I shall have to operate on a student who was sent to me six days after the onset of acute otitis media. He was very ill with influenza previously, and twelve years ago I treated him for bilateral acute otitis media. The old history showed that he had been dizzy for several weeks, and that the trouble had continued in the right ear. When I examined him this time, he said that until this last attack, that ear had been his best ear. Roentgen examination showed nonpneumatization of both mastoids, which possibly was the result of the previous attack of otitis media. We should not ignore

the question as to whether suppuration stops the development of the mastoid and the sinuses, for it has been shown that there is a definite curtailment of development of the sinuses and the mastoids following infection. The patient to whom I have just referred has marked defects of hearing, especially in the right ear, headache and loss of appetite. Those are my indications for operation, especially in the light of the previous history of aural infection.

DR. AUSTIN A. HAYDEN: I think that spinal punctures are absolutely indicated, and I have never seen any harm from them in cases in which there was a suggestion of intracranial involvement. I also think that caloric tests are far more important than the measurement of bone conduction.

A high leukocyte count with a high percentage of polymorphonuclears is an extremely valuable sign in the absence of other inflammations in the body that will account for such a count. I feel also that a Schilling shift to the left is an important factor.

Roentgenograms, even though expensive, are well worth while, and the early picture is of tremendous importance in these cases, perhaps not from what it shows, but from the basis that it establishes for comparison with subsequent pictures that indicate changes in the bone.

In regard to the tenderness being due to periostitis and not to involvement of the bone, it seems to me that, barring the presence of furunculosis and such conditions, it is extremely difficult and quite hazardous to attempt to distinguish between these two conditions.

DR. A. H. ANDREWS: I agree with Dr. Hayden that if roentgenograms are to be of the greatest value they should be taken early and should be repeated as the disease progresses. I further agree that postauricular periostitis originates from conditions within the bone and not from external causes. Periostitis accompanying suppurative otitis is a strong indication for operation on the mastoid.

I was surprised to hear the statement that spinal puncture never does harm. When the patient's condition grows worse after spinal puncture, I know of no way of determining positively what his condition might have been without the puncture. Dizziness is undoubtedly an important symptom suggesting the possibility of labyrinthine involvement.

Dr. Mundt remarked that it is important to know what is happening inside the mastoid. This is true, but it is just as important to know what is happening just outside the mastoid, i. e., in the internal ear, the brain and the lateral sinus. Pain that appears after the patient should be improving is a strong indication for operative intervention.

My method of transillumination is simple. I use a small fountain-pen flashlight. With a speculum pushed rather deep into the auditory canal, and with the room darkened, the flashlight is placed well back on the mastoid. Then the posterior wall of the auditory canal is observed to see if it becomes illuminated. The only light reaching the posterior wall must go through the bone. If the bone is diseased, less light will go through than if it is normal.

The safety of the patient is always the first matter of importance. In his interests, the indications for an operation should be influenced to a considerable extent by the experience and ability of the operator. If the surgeon who is to operate has had little experience or if his experience has not been satisfactory, it is his duty to the patient to wait until operation becomes imperative. To a certain point it is better for the patient to take chances with nature than with an inexperienced surgeon. On the other hand, if the surgeon who is to operate has had wide experience and the results of his operations have been satisfactory, he is justified in operating earlier.

Directory of Otolaryngologic Societies*

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gabriel Tucker, Bronchoscopic Clinic, Hospital of the University of Pennsylvania, Philadelphia.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: New Orleans. Time: 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Ind. Time: Sept. 14-19, 1931.

American Bronchoscopic Society

President: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.
Secretary: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Harry P. Cahill, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

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*Secretaries of societies are requested to furnished the information necessary to make this list complete and to keep it up to date.

Sioux Valley Eye and Ear Academy

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

Southwestern Michigan Triological Society

President: Dr. C. W. Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. F. J. Peirce, 511 N. Main St., Pueblo.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President. Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Douglas Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.

Montana Academy of Oto-Ophthalmology

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

North Dakota Academy of Ophthalmology and Otolaryngology

President: Dr. F. L. Wicks, Valley City.
Sceretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

Oregon Academy of Ophthalmology and Otolaryngology

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, 3117 West Ave., Newport News.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. L. McDougall, 50 Whitehall St., S.W., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. William R. McKenzie, 11 E. Chase St., Baltimore.
Secretary: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. George J. Dennis, 25 E. Washnigton St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. Raymond Goux, 1551 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. John M. Auwerda, 878 President St., Brooklyn.
Secretary-Treasurer: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W.: Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. W. P. Haney, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. James Homer McCready, 121 University Place, Pittsburgh.
Secretary-Treasurer: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 634 N. Grand Blvd., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott Applewhite, 706 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wilber F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

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CONTENTS

PAGES

THE SPECIFIC PROPHYLAXIS OF PNEUMOCOCCUS AND STREPTOCOCCUS MENINGITIS: I. SERUM PROPHYLAXIS. JOHN A. KOLMER, M.D., AND K. W. AMANO, M.D., PHILADELPHIA.. 125

MYOCLONIC MOVEMENTS OF THE LARYNX AND PHARYNX: A MANIFESTATION OF EPIDEMIC ENCEPHALITIS. JOHN H. CHILDREY, M.D., AND HARRY L. PARKER, M.B. (DUBLIN), ROCHESTER, MINN. 139

FATAL HEMORRHAGE RESULTING FROM THE EXTENSION OF A RETROPHARYNGEAL ABSCESS TO THE DEEP VESSELS OF THE NECK. JACOB LIFSCHUTZ, M.D., CHICAGO..... 149

A STUDY OF TINNITUS. R. L. WEGEL, NEW YORK..... 158

THE LARYNX IN PARALYSIS OF THE RECURRENT LARYNGEAL NERVE. JAMES I. FARRELL, M.D., CLEVELAND..... 166

THE SYMPTOM OF VERTIGO. W. E. GROVE, M.D., MILWAUKEE.. 177

PRIMARY TUBERCULOSIS OF THE NASAL MUCOUS MEMBRANE. FRED Z. HAVENS, M.D., ROCHESTER, MINN..... 181

THE PATHOGENESIS OF OTOSCLEROSIS. K. WITTMAACK, M.D., HAMBURG, GERMANY 186

MELANIN IN THE INNER EAR. DOROTHY WOLFF, M.A., ST. LOUIS. 195

NEWS AND COMMENT..... 212

OBITUARY:

ROBERT CLYDE LYNCH, M.D..... 213

ABSTRACTS FROM CURRENT LITERATURE..... 216

SOCIETY TRANSACTIONS:

MASSACHUSETTS EYE AND EAR INFIRMARY OTO-RHINO-LARYNGOLOGICAL SERVICES..... 234

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY..... 238

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES..... 251